THE American Journal OF Gastroenterology

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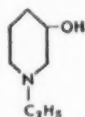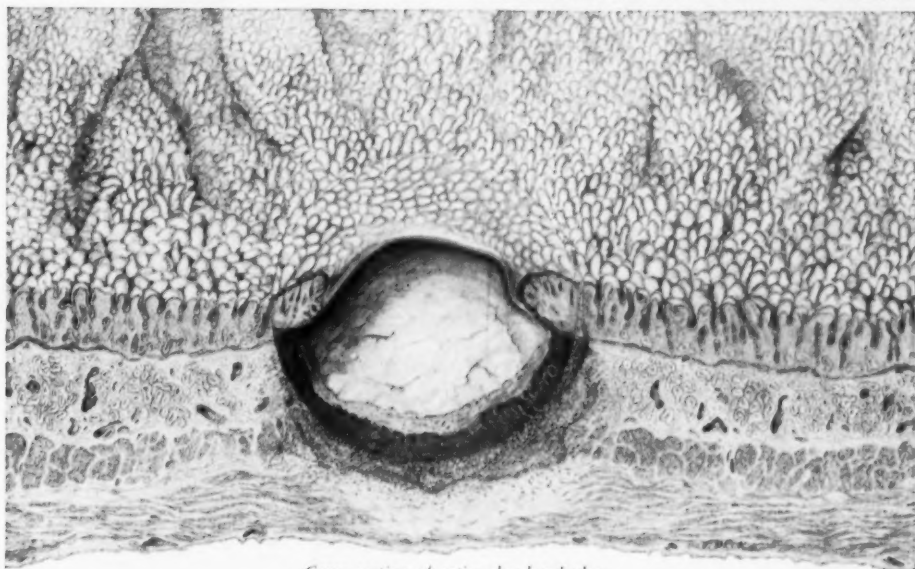
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HIATAL HERNIA, EPIPHRENIC DIVERTICULUM AND CARDIOSPASM*

F. JOHNSON PUTNEY, M.D.

Philadelphia, Pa.

Benign lesions of the lower esophagus present many unique and often puzzling problems in both diagnosis and treatment. Although regarded as benign in nature considerable disability and discomfort to the patient may be produced. It must also be borne in mind that in some cases it is better to render treatment with a view toward relieving symptoms rather than obtaining eradication of the lesion. While anatomic correction of the defect can be accomplished surgically, physiologic return to normalcy does not follow in many instances and the patients may continue to have symptoms referable to the lower esophagus, which at times become quite severe. Since the lower end of the esophagus contains an anatomic and physiologic pinch cock, which remains closed in the normal state and only opens to allow the progress of food from the gullet into the stomach, maintenance of this relationship is essential to proper functioning. After surgical interference with this mechanism restores anatomic continuity on the roentgenogram and re-examination shortly thereafter reveals a normal esophagus, the patient is considered cured. It has been my experience, however, that when the hiatus has been disrupted these patients experience reflux esophagitis with regurgitation and pain, and if they are examined several years later, the esophagus is generally found to be dilated, the seat of severe esophagitis and the previous postoperative improvement is replaced by the original symptoms of equal or more marked severity. Holinger et al[2] have also demonstrated an increasing number of strictures following esophageal surgery, and these now constitute the single largest source of esophageal strictures.

ESOPHAGEAL HIATAL HERNIA

Most reports of large numbers of cases of diaphragmatic hernia agree that esophageal hiatal hernia is the commonest type of the nontraumatic group. Acquired herniae are being recognized more frequently now as the cause of

213

symptoms, and whereas formerly a small hiatal hernia was noticed on the roentgenogram it was sometimes passed off as being of no significance when it was the source of the difficulty.

There are two types of esophageal hiatal hernia: 1. paraesophageal hernia in which the lower end of the esophagus remains fixed in its normal position and a portion of the stomach herniates through the hiatal ring adjacent to the esophagus; 2. the true sliding hernia in which there is protrusion of both the lower end of the esophagus and the upper end of the stomach into the thorax. While this distinction is of academic interest only it is of practical importance to differentiate an acquired esophageal hernia from a congenitally short
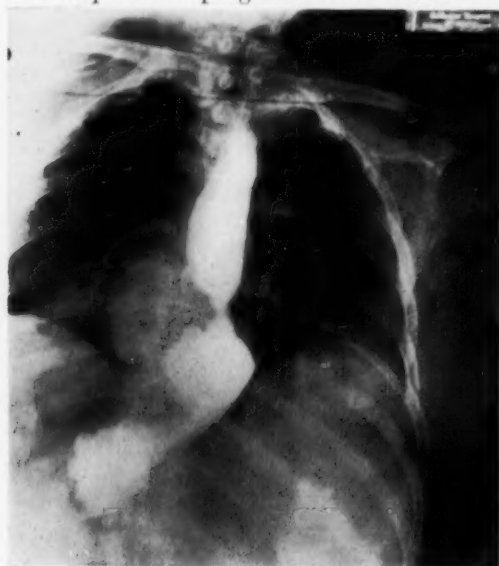
Fig. 1—A 55-year old female had symptoms referable to the upper gastrointestinal tract. Cholecystectomy with removal of gallstones failed to relieve the symptoms. Subsequent roentgen studies revealed a large hiatal hernia which was repaired surgically and the symptoms disappeared.

esophagus. In the latter condition there is not sufficient esophageal length to allow the gastric pouch to reach the diaphragm, so that the stomach occupies its embryonic thoracic position and has never gravitated to the abdominal cavity. The length of the esophagus affords the main difference between esophageal hiatal hernia and congenital short esophagus. This assumes additional importance in evaluating a patient for surgical treatment, for it is possible to repair an esophageal hiatal hernia and replace the stomach in the abdominal cavity, but in congenitally short esophagus the stomach cannot be returned to the abdominal cavity because of the short length of the esophagus and any attempt at surgical

treatment makes the condition worse. Medical measures and esophagoscopic dilatations of stenosis occurring in a congenitally short esophagus are superior to any surgical attempt at correction.

The amount of mechanical interference with the herniated stomach, the degree of diaphragmatic dysfunction, and the amount of increased intrathoracic pressure determine the type and number of complaints. Benign lesions of the lower esophagus have many symptoms in common and these vary widely and usually are numerous. This is especially true in hiatal hernia where the complaints may suggest peptic ulcer, coronary occlusion, intestinal obstruction, cholecystitis
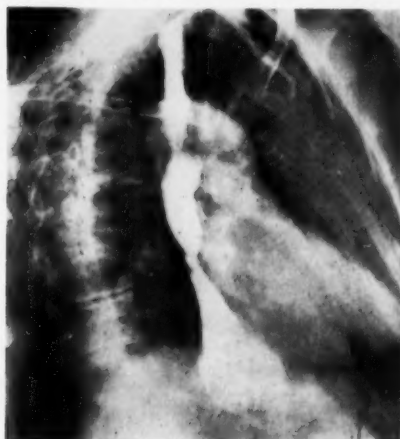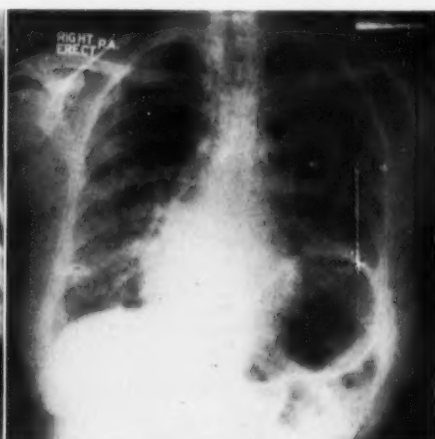
Fig. 2   Fig. 3

Fig. 2—In contrast to an acquired hiatal hernia a congenital short esophagus lacks the mucosal redundancy and the esophagus is stretched and tense. Stenosis at the esophagogastric junction in the chest is a frequent finding, and ulceration may develop. The treatment consists of dilatation and local applications to the ulcerations.

Fig. 3—Eventration of the diaphragm may be confused with hiatal hernia. In this case of eventration of the left diaphragm the symptoms were indefinite and similar to those of hiatal hernia. It is distinguished by the presence of the gastric air bubble below the diaphragm, absence of lung tissue behind the air pocket, and paradoxical movement of the involved diaphragmatic leaf.

or other diseases. The clinical diagnosis is difficult if dependency is placed on subjective manifestations alone and thorough roentgen and esophagoscopic study is necessary to make the correct diagnosis.

In a study of 110 patients with esophageal hiatal hernia by Clerf et al[5] approximately one-half presented the outstanding primary symptom of dysphagia, followed closely by pain in some locality. The association of dysphagia and pain was so frequent that when these two symptoms occur together the diagnosis of esophageal hiatal hernia should be strongly suspected. While the difficulty in swallowing was present for liquids and solids alike, solid foods usually gave

the most distress. Curiously, some of the patients referred their difficulty to the neck region when in reality the disturbance was at the lower end of the esophagus.

The extent, character and location of the pain was quite variable. In many cases it was located in the chest either precordial or radiating upward into the shoulder or downward into the abdomen. The pain was not constant but came in attacks which varied in duration from a few minutes to hours. The attacks were mild and spaced at frequent intervals in the early stages but became more constant and severe after the condition had existed for a period of time. In other instances the intake of food initiated an attack of epigastric pain which was relieved at times by belching or vomiting. Four patients in this series were treated for coronary thrombosis because of the severe radiating precordial pain.

The chief difficulty in diagnosis occurs when one or more conditions coexist, and it becomes necessary to determine which lesion is actually producing the symptoms. In this series one patient had a hiatal hernia, cholecystitis and coronary insufficiency while in two others the hernia was complicated by peptic ulcer. Another patient had her gallbladder which contained stones removed for digestive disturbances and one year later, after the symptoms persisted, an esophageal study was made and the presence of a hiatal hernia discovered. After surgical repair of the hiatal hernia the symptoms disappeared indicating that they were probably all due to the hernia rather than the gallstones.

On roentgen examination the finding of gastric rugae above the diaphragm clinches the diagnosis, and while the pinch cock appearance at the hiatus is often absent, the esophagogastric junction can usually be distinguished and found above the diaphragm level. It must be realized that some herniae are reducible so that it may be necessary to view the lower end of the esophagus and the stomach from many angles to determine their relationship, and in some instances the intraabdominal pressure must be increased sufficiently to force the stomach into the thoracic cavity. Eventration of the diaphragm may be confused with hiatal hernia but in this condition no lung tissue can be visualized through the gas bubble which may be thought to be in the thoracic cavity, no abdominal viscera can be demonstrated above the diaphragm and the diaphragmatic movements are paradoxical.

The esophagoscopic diagnosis is made on 1. anatomic demonstration of the stomach above the diaphragm, 2. relaxation of the esophageal mucosa and 3. absence of the normal pinch cock action at the hiatus. Cardioesophageal relaxation, even though there is no actual herniation, probably represents the first stage in the formation of the hiatal hernia, and may give the same symptoms as an actual hernia. In some instances, narrowing at the esophagogastric junction is observed and this frequently follows the healing process of ulceration. Ulcerations, varying from a small area at the point of stenosis to extensive change covering the entire stenotic area, may be found. They may give rise to frequent hemorrhages some of which become so severe as to necessitate frequent blood transfusions and surgical treatment to relieve this complication.

The majority of patients (69 per cent) in this series were relieved symptomatically by medical or mechanical treatment or both. Usually, the smaller herniae caused less distress, but some of the very small herniae were symptomatic, did not respond to conservative methods and required surgical treatment. The conservative treatment is directed toward providing an adequate passageway and the relief of distressing symptoms. When mechanical treatment is necessary to enlarge the lumen it can be done by dilatation. The frequency of dilatation depends considerably on the type and severity of the constriction and in some

Fig. 4   Fig. 5

Fig. 4—This 7-year old boy with achalasia had 52 dilatations over a period of seven years with little improvement. Relief followed the surgical procedure of Heller's esophago-cardiomyotomy.
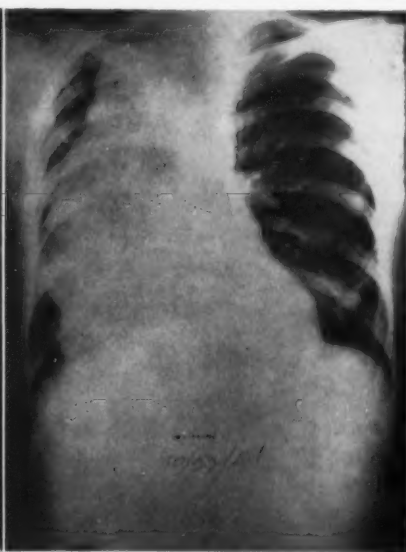
Fig. 5—In this case of achalasia the esophagus occupied practically the entire right thoracic cavity and, when filled with food, simulated mediastinal enlargement. Dilatation afforded only partial relief and surgical treatment was needed.

cases may only require treatment two or three times a year. The topical treatment of ulceration may produce healing, but these sometimes return with dietary indiscretions or failure to observe simple medical principles. Medical treatment, consisting of regulation of diet, the taking of liquids with meals to aid in washing down food, together with postural measures to encourage the passage of food are needed. Reclining after meals proved detrimental and at times was followed by a feeling of epigastric fullness. Antispasmodic and antacid drugs, when used to relieve pain, afforded relief to many patients.

The disadvantage of the conservative method of therapy is that it only attempts to alleviate symptoms and has to be continued for a long time. Surgery on the other hand aims at replacing the stomach in the abdomen with repair of the relaxed hiatal ring and relief of symptoms without further treatment. Phrenic exeresis alone has provided symptomatic relief in many cases, particularly those in the older age group, which are not suitable for surgical repair of the hernia. In this series, 31 per cent were advised to have some form of surgical treatment but of this group only 15 per cent consented to operation. Half of these patients who were poor operative risks had phrenic exeresis alone. Fortunately the recurrences following surgical repair are not numerous and Hedblom[1] reported about 5 per cent recurrences in all types of operated diaphragmatic herniae. The definite indications for surgical repair are: 1. progression of symptoms in patients in good physical condition; 2. severe and recurrent hemorrhage from chronic or recurrent ulceration either at the lower end of the esophagus or in the gastric pouch and 3. incarceration or threatened strangulation.

Epiphrenic Esophageal Diverticulum

The epiphrenic esophageal diverticulum found at the lower end of the esophagus is pulsion in type and commonly is located within a few inches of the hiatus. While it does not occur frequently and the cause has not yet been determined the pouch projects more often into the right thoracic cavity than the left. This type of diverticulum is caused by pressure from within and is essentially a herniation of the mucous membrane through the muscle coats. In contrast to the pulsion type, the traction diverticulum, which rarely produces clinical symptoms, is due to contraction from fibrosis caused by an inflammatory process of the outer esophageal wall associated with disease of the lymph nodes at the tracheal bifurcation and hilar region that have become adherent to the esophagus.

In the cases reported by Putney and Clerf[4] the percentage of surgically treated epiphrenic diverticula as compared to all types of pharyngeal and esophageal diverticula was about 4 per cent. On histologic section it was shown that all layers of the esophagus were present but the muscle coat was thin and sparse with consequent weakness of these fibers.

Supradiaphragmatic pulsion diverticulum presents no characteristic symptoms, and the symptoms are similar to those of other benign lower esophageal lesions. The symptoms progress with an increase in size of the sac which may become large enough to produce pressure and distortion of the subdiverticular esophagus with resulting obstruction. Dysphagia, epigastric distress, regurgitation, anorexia, nausea, blood spitting and cough were observed in these lesions.

The roentgenographic picture of epiphrenic diverticulum may be confused with achalasia in which there is a large sigmoidal dilatation of the distal esophagus. Carcinoma of the lower esophagus may produce a false diverticulum above

the carcinomatous stenosis but generally there is a uniform sacular dilatation in which the entire circumference participates.

Although the diagnosis is commonly made by roentgenologic study, esophagoscopy serves to confirm the diagnosis and detect complicating lesions, at the same time providing additional information about the mucosal changes. The possibility of malignancy in the fundus of the sac must always be considered and four cases of epiphrenic diverticulum associated with carcinoma in the sac

Fig. 6—Roentgenogram of a 62-year old male diagnosed as achalasia. Esophagoscopic examination revealed a narrow, firm hiatus and entrance into the stomach could not be accomplished. At operation carcinoma of the gastric fundus extending submucosally up the lower end of the esophagus was found. Infiltration of the lower esophagus from carcinoma originating in the stomach was difficult to diagnose in the absence of ulceration, but esophagoscopy is a valuable aid.

have been reported. Investigation of the subdiverticular esophagus is essential, and when there are associated lower esophageal lesions these may escape detection because of distortion incident to the diverticulum. The diverticula have large mouths and the proximal lumen leads directly into the dilatation while the distal esophageal lumen may be narrow, distorted and pushed from its normal location. When the site of the diverticulum is located and it can be ascertained to which side the fundus projects, the surgeon receives valuable aid in selecting the most

accessible surgical approach for exposure. When surgical treatment is contemplated the presence of retained food should be eliminated prior to surgery.

If the diverticulum is asymptomatic no treatment is required. One patient who experienced substernal pain upon taking food declined surgical treatment and during the past 18 years has been carrying on his usual activities without undue distress provided the diet is watched. The patient has found that it is necessary to rigidly control the diet, for as soon as he overloads the diverticular pouch, or eats coarse food not thoroughly chewed pain develops.

As the diverticulum enlarges it retains food and produces secondary esophageal obstruction resulting in insufficient food intake and nourishment, and commonly surgical treatment is needed. The operation of choice is simple excision of the sac. When several lesions coexist and surgical treatment is employed it seems best to treat each lesion individually. In a case that I observed with a hiatal hernia and achalasia in association with the diverticulum the sac was surgically excised, the hernia was repaired with replacement of the stomach in the abdominal cavity and Heller's esophagocardiomyotomy for achalasia was performed.

CARDIOSPASM

Cardiospasm or achalasia may begin at any age and occurs in infancy and childhood oftener than is generally believed. In a series of 286 cases at Jefferson Hospital the youngest patient was 13 days and there were infants of 4, 18 and 19 months of age. Two patients aged 22 and 53 dated their symptoms from birth.

Of the total number the onset of the disease was sudden in 24 per cent and insidious in the remaining 76 per cent. Although no special effort was made in taking the histories to elicit past or present mental disturbances in those patients with sudden onset of symptoms the initiating circumstances revealed psychogenic factors dating to a specific incident in 62 per cent.

Because of the lack of attachment posteriorly, the terminal esophagus moves up and down with diaphragmatic movement for approximately 1½ inches. The terminal end twists to the left and as the diaphragm moves upward during expiration, the right wall of the esophagus pouches downward and it is at this point that cardiospasm begins. The twisting of the lower end of the esophagus upon itself from right to left at the crus changes the diameter from transverse to anterior-posterior.

Much of our knowledge of the detailed anatomy of the esophagus has been contributed by Mosher[3] who demonstrated sizeable glands between the crura and esophagus, both in children and adults, and a connective tissue sheath which is continuous without break with the lesser omentum. In the lower half of the esophagus Auerbach's plexus of sympathetic nerves with large ganglia between the circular and longitudinal muscle fibers is also found, and some observers feel that fibrosis of this plexus with the resulting functional loss of ganglion cells is the cause of cardiospasm. On the other hand Mosher has pathologic evidence

to support his contention that the etiology is fibrosis of the terminal portion of the esophagus in the crural canal. Vinson[6] believes the cause may be a degenerative change in the vagus nerve fibers innervating the esophagus. While all of these diverse opinions have some substantiation, the etiology remains unsettled.

The essential lesion is a cylindrical narrowing of the terminal esophagus at the crural ring. The back pressure in the esophagus causes hyperplasia of the musculature involving one or all of the layers. At first the hyperplasia is confined to the region just above the narrowing but later extends up the esophagus for varying distances resulting in a loss of muscular power.

Fig. 7   Fig. 8

Fig. 7—Large epiphrenic pulsion diverticulum with retained food producing constriction of the subdiverticular esophagus. Surgical excision was followed by relief of symptoms.
Fig. 8—This 36-year old female had an achalasia and hiatal hernia in addition to the epiphrenic diverticulum. Recovery followed surgical treatment which consisted of excision of the sac, repair of the hernia and esophagocardiomyotomy.

The symptoms usually exist for a long period of time and errors in diagnosis are frequent. The most common erroneous diagnosis is nerves with esophageal stenosis and indigestion also frequent. Bizarre diagnoses such as thyroid disease, arthritis, tuberculosis and renal disease have been encountered.

In evaluating the symptoms in this series a majority of the patients had complaints similar to that of other lower esophageal diseases but the most common was dysphagia followed by regurgitation, weight loss and pain. While respiratory symptoms caused by aspiration of esophageal contents or pressure

from the dilated esophagus occurred, they were not common and consisted of cough, dyspnea, wheeze, hiccough and hoarseness. In the majority of patients there was dysphagia for fluids and solid foods alike, although difficulty with cold liquids was the initial disturbance in many. Generally, any fluid or food caused a sensation of obstruction, but many patients reported the greatest difficulty from raw apples and ice cream.

At first the dysphagia was intermittent but as the condition progressed it became continuous although the degree of severity would vary from day to day. Complete obstruction to the passage of food into the stomach for days was a common occurrence. The sensation of obstruction was generally referred to the epigastrium but often the patient localized the site of food lodgement to the upper esophagus. Any emotional stress increased the dysphagia in practically all of the patients.

Regurgitation occurred immediately following the ingestion of a meal or was delayed for an indefinite time depending upon the amount of esophageal dilatation above the obstruction. When food was retained in the esophagus for a long time various degrees of fermentation resulted. The material regurgitated consisted of undigested food in various states of decomposition which presents a characteristic appearance. Retention of food initiated regurgitation and when small amounts of food could get into the stomach the regurgitation was less marked for the esophagus became slowly decompressed. If there was ulceration of the mucous membrane small amounts of blood were often present in the regurgitated material, but frank hemorrhage was infrequent and not severe enough to require treatment. The loss of weight was attributed to failure of ingested food to reach that portion of the gastrointestinal tract through which absorption could take place.

Since physical examination as well as laboratory study revealed little, the diagnosis depended upon roentgenography and esophagoscopy. Carcinoma of the gastric cardia with the growth extending into the hiatus esophagus simulates cardiospasm. This is an extremely difficult distinction to make both by roentgenography and esophagoscopy for the narrowing may be smooth, nonulcerated and resemble cardiospasm. When the growth has not broken through the mucous membrane the only significant finding is the rigidity of the narrowed portion in which there is absence of peristaltic movements and inability to introduce the esophagoscope into the stomach. In uncomplicated cardiospasm peristalsis, while decreased, is still present, and no abnormal constriction is noted on passage of the esophagoscope into the stomach.

In the majority of patients mechanical dilatation of the esophageal hiatus gave the best result. There are several methods of stretching the hiatus and it matters little which method is used provided it is safe and adequate dilatation can be accomplished. In performing dilatation it is essential that the muscle fibers at the hiatus be stretched and even avulsed. I prefer the method of

aerostatic dilatation with the dilator passed over a previously swallowed string. This can be accomplished by passing the dilating bag into the stomach so that approximately a half rests in the stomach and the other half in the esophagus. The maximum pressure exerted is 20 mm. of air and the pressure is never increased beyond the point at which the patient complains of definite pain. In my experience, except for dietary measures, medical treatment including various drugs as antispasmodics, vitamins, aminophyllin, nitroglycerin and others has afforded little relief. Since psychic trauma is a factor in the initial phase of many cases, psychotherapy is useful in the first few weeks, but once the disease has fully developed with dilatation and retention psychotherapy is useless. To be beneficial it must be employed before the esophageal walls have become thick and leathery from hyperplasia and the changes have become irreversible.

When dilatation is employed prompt relief should be obtained. If there is no improvement after several dilatations there is no value in continuing this form of treatment. In those cases in which the cardiospasm is long standing and the walls of the esophagus greatly hyperplastic with a tortuous and sigmoidal dilatation surgical treatment is indicated. In these cases the conservative surgical approach using the Heller esophagocardiomyotomy procedure has given the best results. By splitting the muscle fibers and leaving the hiatus intact postoperative complications are minimal and reflux esophagitis diminished while at the same time the constriction is overcome.

SUMMARY

In reviewing esophageal hiatal hernia, epiphrenic diverticulum and cardiospasm the symptoms are similar, and the diagnosis depends upon thorough roentgenological and esophagoscopic study. While other lesions may be simulated the chief diagnostic difficulty occurs when two or more conditions exist, and it is necessary to determine which disease is actually responsible for the symptoms. Surgical treatment is necessary in many instances, but the conservative management also has a place in treatment. The importance of maintaining the esophagogastric junction and hiatus intact after surgical treatment is essential to prevent recurrence of disabling symptoms, some of which may become more severe than the original ones.

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CONSERVATIVE MANAGEMENT OF OCCLUSIVE DISEASES OF THE ESOPHAGUS*†

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The relative impunity with which the esophagus can now be attacked surgically[1] presents a challenge to those methods of treatment which are non-surgical. Admittedly carcinoma, being a disease requiring extirpation, cannot be treated conservatively. There are, however, many stenosing conditions—both functional and organic which lend themselves readily to conservative management. Some of these conditions can be treated by physicians without special training and with knowledge of but a few essential facts pertaining to the esophagus.

ANATOMICAL CONSIDERATIONS:

The esophagus lies on the dorsal column and, therefore, follows its antero-posterior convexity. Just above the diaphragm it angulates obtusely forward
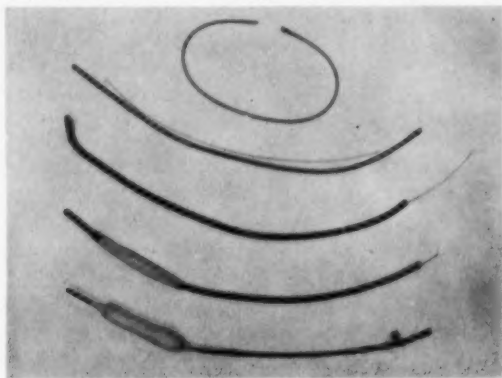
Fig. 1–Dilators. From top to bottom; nasal tube; 22 F. colon tube with wire guide; Ewald tube with wire guide inserted and showing finger-tip; balloon dilator not inflated; balloon dilator inflated.

and to the left as it passes through the diaphragm. This obtuse angulation represents the site of greatest hazard from leakage or rupture upon manipulation. Whereas previously rupture of the esophagus spelled almost certain death, the advent of antibiotics has greatly reduced this danger. The esophagus is

not covered with serosa such as is found in the rest of the gastrointestinal tract. This serosa elsewhere acts as a tough limiting membrane reducing the danger of rupture and spread of infection. In the esophagus there is danger, although remote, not only of causing rupture by the passage of a stomach tube, but of irritating the esophagus so as to cause a chronic esophagitis with resulting stenosis. The measurement of the length of the esophagus and the localization of the site or sites of stenosis can best be made from the upper central incisors. In the adult the length of the esophagus is remarkably uniform, from 38-40 cm.[2,3]. The site of obstruction is easily determined. In the first place, the patient can readily indicate the exact level of obstruction by pointing to the level of

Fig. 2—Balloon dilator inflated *in situ*.

distress. In cardiospasm he will point to the region of the ensiform cartilage. If the obstruction is in the mid-esophagus, he will be able to locate it substernally at whatever level it occurs. If the esophagus is not dilated, a soft rubber tube may be compared to the extension of an index finger for palpating the type of obstruction and indicating its level by measurement from the central incisors. A 22 F. colon tube is a convenient exploring device for getting information. A nasal tube, such as the Wangensteen or Levin tube may be too soft and pliable to transmit tactile sense. The Ewald tube may serve as a convenient substitute for the 22 F. colon tube. The undilated esophagus acts as

a supporting sheath to prevent the buckling of an exploring soft rubber tube. If the esophagus is dilated, a semirigid wire curved to the convexity of the dorsal spine and inserted in the rubber tube can be used. The wire is inserted to within two inches of the tip of the rubber tube. This rubber tip acts as a guide for the tube and protects the esophagus similar to the "finger-tip" guide of the gastroscope[3].

In benign stenosing diseases of the esophagus the problem is usually simply that of effecting adequate dilatation to enable the esophagus to fulfill its only function of conveying an adequate amount of food from the mouth through the chest into the stomach. The principles of therapy involved in correcting stenoses vary with the pathogenesis of the underlying diseases. These may be divided into: 1. functional occlusions without organic change, 2. functional occlusions
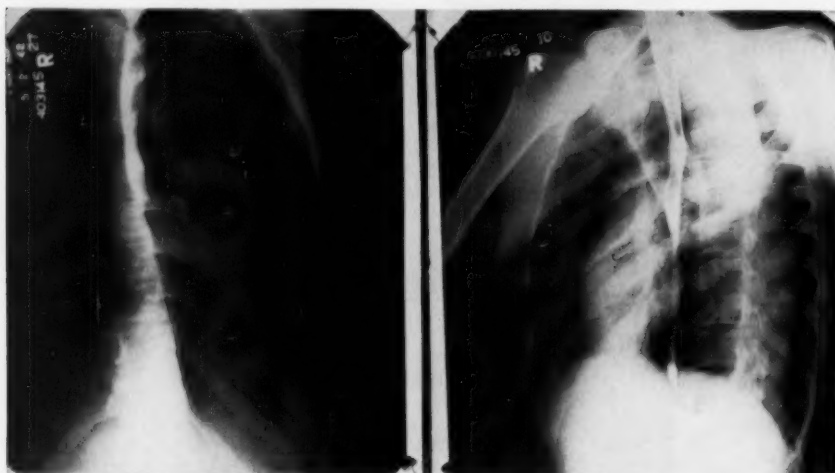
Fig. 3—Chronic esophageal stenosis in 18-year old male patient occluded by a piece of meat and relieved by passage of soft rubber tube.

with organic change such as cardiospasm and, 3. chronic stenosing esophagitis, which occurs most frequently following infection, peptic ulcer of the esophagus and chemical esophagitis.

The easiest type of occlusion to treat is that of functional disturbance without organic change and which may or may not be associated with psychosomatic disturbance. This is the only type in which drug therapy may be effective. It is probable that most of us at some time have experienced a transient localized occlusion not recognized as such. This occurs usually while eating and manifests itself as a transient substernal distress. Relief is usually obtained by the drinking of some fluid. Such instances do not come to the physician for management. In other instances, such as might be represented by the

Plummer-Vinson syndrome[4], dysphagia may be corrected by the passage of an Ewald tube.

Where actual dilatation of the lumen exists, the technic of treatment will obviously vary with the nature of the pathology. If the condition is looked upon as being functional, as in cardiospasm, an expansile type of balloon dilator, such as the Plummer dilator[5], must be used. If the stenosis is due to postinflammatory scar tissue constriction, a rigid type of graduated dilator is needed. Graduated metal olives are ideal.
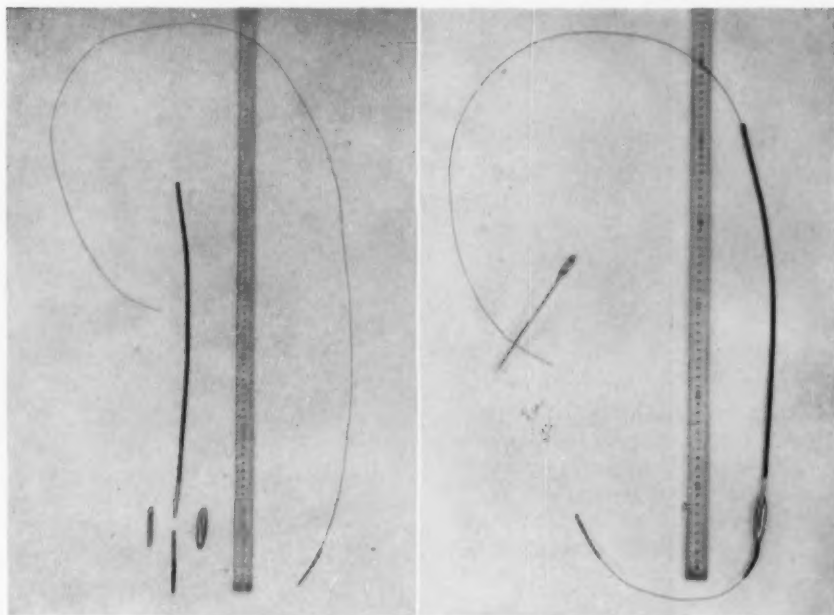
Fig. 4—Rigid dilator assembled and disassembled.

Cardiospasm, as it is encountered clinically, may be looked upon as being both a functional and an organic esophageal derangement. This may be defined as a continuous functional spasm at the cardiac end of the esophagus with secondary organic change manifesting itself as a permanent dilatation through-out the esophagus above the site of spasm. The esophagus becomes a reservoir. With esophageal stasis, inflammation, cicatrization, and finally organic stenosis develop whereas in the beginning the change was only that of spasm. The treatment of the spastic condition is the dilatation of the obstruction with an elastic balloon-type of dilator. Various satisfactory technics of treatment have been described. The method used for the past 22 years at the State of Wisconsin General Hospital is a modification of that popularized by Plummer[5].

THE BALLOON DILATOR

A 22 F. colon tube or an Ewald tube are used. A grooved brass plug is driven to within three inches from the tip of the tube. This seals the tube and forms an anchor for the distal end of the balloon. A brass spool is then driven to within ten inches of the tip of the tube to form the proximal anchor for the balloon and provides for the passage of the previously described guide wire. Multiple perforations are made between the two anchors so that fluid can flow into the balloon. The balloon itself consists of three layers. The outer layer is of Penfield surgical tubing. The middle layer is silk to provide strength

Fig. 5—Chronic postoperative stenosing esophagitis in a 61-year old male requiring dilatation in 1939, symptom-free since. Roentgenogram of February 7, 1939.

and to give the balloon size and shape. The diameter of the balloon bag is 2½ inches. The inside of the balloon bag is lined with condom rubber to act as a seal. These materials for manufacture and repair of this dilator are readily available in the average hospital.

Because the esophagus is dilated, an unsupported rubber tube would only curl up in the esophagus if one attempted to try to pass such a tube as is used for gastric analysis. A semirigid guide wire, therefore, bent to conform to the curve of the dorsal spine is used as an obturator in a soft rubber tube. It is inserted to within about two or three inches of the end of the tube so as to

leave the end of the tube introduced into the esophagus as a flexible tip. Therein lies the factor of safety. This tip has been referred to by Schindler in his description of the gastroscope as a "flexible finger-tip"[3]. In most instances, this flexible finger-tip will act as a safe guide for the introduction of the rubber tube into the stomach through the area of obstruction. With a spastic type of obstruction and without organic change, no resistance is encountered. When one has been able to introduce such a rubber tube into the stomach it should then be possible to pass a similar tube, over which a dilating bag has been built. Swallowing of a guiding string is thereby obviated. After the dilator

Fig. 6—Same patient as figure 5. Roentgenogram of July 6, 1953 at age of 75 years.

has been introduced the guide can be withdrawn so that the patient may flex his head and be free to move. By means of water[6] or air pressure, the bag may then be distended with safety to the point of the patient's pain tolerance. It is important that the patient receive no preparatory medication, so that pain may be used to determine the amount of pressure tolerated. Complete symptomatic relief is usually effected following a single dilatation. In a minority of cases, there are recurrences. Dilatation may then be repeated. With complete symptomatic relief there is, however, no demonstrable organic change. The patient usually is able to eat all foods immediately after a single treatment. As is well known, the dilatation of the esophagus usually is permanent.

Progress of the patient is determined by the clinical course and not by x-ray progress study, for there will be no change in the appearance of the esophagus
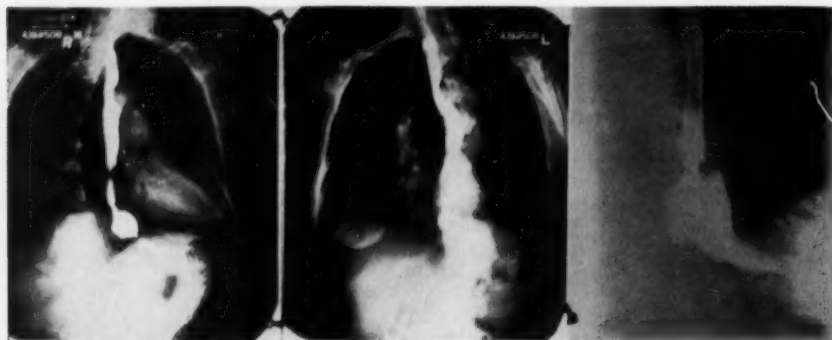
Fig. 7—Peptic ulcer in a short esophagus in a 69-year old female patient with hiatal hernia.

following treatment, even though there is complete symptomatic relief. Failure may be anticipated in the case of long-standing in which organic stenosis at the lower end of the esophagus has replaced functional spasm. Such change

Fig. 8—Postirradiation stenosis of the upper esophagus in a 57-year old female patient. Patient dilated for eight years after irradiation for carcinoma of the esophagus.

may be suspected with the demonstration by means of x-ray of an elongation of the area of occlusion.

THE RIGID DILATOR

The same principles of therapy, with modifications, apply to single or multiple areas of chronic fibrotic stenosis of the esophagus. In these cases, stenosis may be of such high degree that the esophagus cannot be catheterized by even the smallest rubber tube. Complete occlusion is almost never encountered. Any instrumentation must be over a guide. Although direct visualization in any endoscopic procedure is an established ideal, esophageal dilatation cannot be effected adequately by direct visualization. Some type of guide, therefore, is necessary as a protection against perforation. A swallowed string or a fine
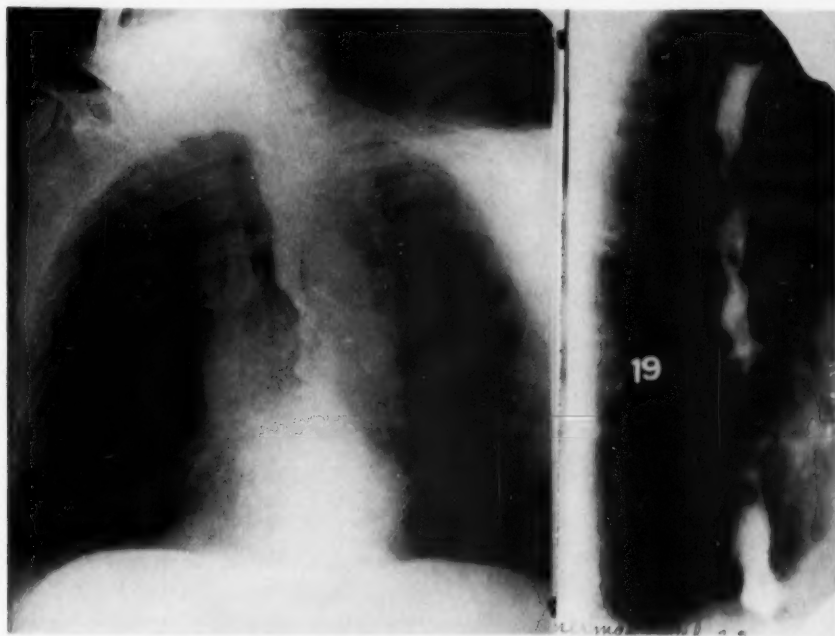
Fig. 9—Stenosis of the mid-esophagus in a 74-year old male patient after irradiation for carcinoma.

wire with a flexible finger-tip provides the needs. We have used, therefore, fine piano wire, to the end of which we have built a finger-tip in the form of a slender, flexible string with a solid tip having a length of three inches and a diameter of two millimeters. Again, without premedication the "finger-tip" guides the wire through the stenosed esophagus. Sufficient length is introduced so that the surplus of wire is curled into the stomach. With such a wire guide in place, the projecting end of the wire can be anchored to a suitable object, usually the operator's shoulder, while graduated metal olives are passed on successive occasions through the stenotic areas. The olives, perforated at both

ends, are carried on a two-piece flexible support. The distal end of the support consists of a short tapered spring attached to a rigid copper tube with a female screw thread. The proximal part of the support is a longer flexible spring attached to another short piece of rigid copper tube with a male screw thread. The olive is kept in place on the rigid tubing when assembled. The proximal flexible spring must be long enough to extend throughout the length of the esophagus. Forty-three centimeters above the olive is a convenient length

As long as the smallest opening can be maintained, a patient may be nourished adequately. In contradistinction to a case with a functional obstruction, the patient with organic obstruction can be rendered symptom-free with relatively little dilatation for the element of spasm is absent. Beginning with the smallest sized dilator, one may attempt dilatation to the extent of F. 54 or even larger. Because scar tissue contracts, retreatment upon subsequent occasions may be necessary following a series of initial treatments. In time, the rigid esophagus usually becomes stabilized, so that further treatment may be unnecessary over indefinitely long periods of time. Again, progress is measured by the clinical course of the patient and not by the appearance of the x-ray film.

SUMMARY AND CONCLUSIONS

1. Management of obstructing benign disturbances of the esophagus is discussed.

2. With the knowledge of a few essential peculiarities of the esophagus many cases now subjected to surgery may be treated by conservative measures.

3. Simple technics obviating the use of a twisted string guide are described.

4. The advantage of a "finger-tip" guide are presented.

5. A modified balloon dilator and a new method for the introduction of dilating olives are described.

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HIATAL HERNIA*

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The relative frequency and clinical importance of esophageal hiatal herniae are not generally appreciated. The diagnosis of hiatal hernia is often not made and, moreover, erroneous treatment for other upper abdominal disease may be instituted when the true offender is a hernia of the esophageal hiatus. The several types of esophageal hiatus herniae perhaps rank only second to peptic ulcer in producing symptoms referable to the upper abdomen.

Esophageal hiatus herniae may be divided into the sliding or short esophagus type and the paraesophageal. There are certain symptoms fairly characteristic of each type but it should be emphasized that there is overlapping in clinical manifestations of both varieties. From a practical standpoint this is not of great importance because if the lesion is symptomatic the proper treatment is surgical intervention.

In the paraesophageal variety of hernia the esophagus enters the stomach at its normal location and at a proper angle below the diaphragm. The esophagogastric sphincter functions and the stomach herniates through the hiatus into the thorax with the cardia uppermost. In this variety of hernia the most characteristic symptom results from the incarcerated cardia and sometimes incomplete gastric obstruction. Manifestations of pulmonary and cardiac pressure may predominate. Regurgitation of acid gastric juice is not excessive because the sphincter or pinch cock action at the esophagogastric junction still functions.

The second variety of esophageal hiatal hernia, the sliding or short esophagus type is much more frequent. It occurs commonly in older people and especially in females. This lesion is characterized by an elevated, shortened esophagus which may enter the cardia of the stomach at a less acute or even as much as a 90 degree angle above the diaphragm. The cardia of the stomach and the abdominal esophagus represents the herniated elements. Usually the esophagogastric junction does not function and, accordingly, there are varying degrees of reflux of gastric juice. The clinical findings, therefore, are most apt to reflect the acid peptic action on the esophagus. If peptic digestion of the esophagus continues it may be followed by ulceration, fibrosis and shortening of the organ. In extreme cases there will be stricture formation. When these complications of a sliding or short esophagus type of hiatal hernia occur, the lesion becomes serious and irreversible.

Hiatal herniae may include a defect extending from the aortic hiatus to the esophageal hiatus with the lesion situated in the posterior mediastinum. These have been called esophagoaortal herniae by Adams[1]. The anatomic position accounts for the high incidence of cardiorespiratory symptoms produced by these lesions.

Congenital short esophagus may be confused with sliding hiatal hernia but more often the converse is true, and the shortened esophagus associated with esophagitis from gastric regurgitation is thought to be congenital in nature.

Actually the true congenital short esophagus is rather rare. Distribution between men and women is about the same and symptoms become evident at a much younger age. In the congenital lesions the cardia of the stomach is retained and rotated above the diaphragm by the short esophagus. Usually the esophagogastric junction is patulous. Symptoms are produced by the accompanying esophagitis and ulceration.

The most frequent symptom of esophageal hiatus hernia is intermittent gastric distress ranging from mild dyspepsia to incapacitating pain. The pain is usually located in the epigastrium or beneath the lower sternum. If there is radiation to the shoulder and down the arm an erroneous impression of coronary disease may be gained. These symptoms usually occur during and immediately after meals. It is aggravated in the reclining or recumbent position and often relieved by change to an erect sitting or standing position. If, however, the stomach is permanently incarcerated the pain may be continuous and not relieved by postural changes.

It is important to mention that in lesions producing pain mocking cardiac disease, electrocardiographic changes may sometimes be present which suggest coronary occlusion. These changes in the electrocardiogram, however, usually revert to normal configurations quickly and the leucocyte count, sedimentation rate and temperature are not compatible with coronary occlusion. Moreover, the pain is usually relieved by the erect position and brought on by meals. Occasionally a real diagnostic dilemma exists in patients in whom both coronary disease and hiatal hernia are present. This situation is more frequent in the older age group.

Since patients with hiatal hernia are troubled with bloating, belching, a sensation of epigastric obstruction, regurgitation and vomiting, it is easy to confuse symptoms related to the hernia with gallbladder disease and diseases of the stomach and pancreas.

Patients with hiatal hernia are often thought to have duodenal ulcers, since they develop their own treatment when they find that taking alkalis and drinking milk will relieve the distress. If this pain is relieved by postural changes and there is no evidence of duodenal or gastric ulcer on roentgen examination, a hiatal hernia is likely to be the cause of the symptoms.

Continued bleeding from ulcerations may result in hematemesis or insidious loss of blood in the stool. Resultant anemia may be difficult to diagnose but if present and hypochromic and macrocytic in nature, in the absence of other cause, a hiatal hernia should be considered.

Sometimes cough, dyspnea and even asthmatic-like attacks may follow meals as a reflex or pressure phenomenon from the hernia. Cholelithiasis, diverticula of the colon, ulcers and diverticula can be confused with or coexist with a hiatal hernia and each element must be carefully evaluated before treatment is recommended. Usually hiatal herniae cause a chronic, progressive pattern of symptoms. If, however, gastric strangulation or hemorrhage occurs, the onset may be dramatic. Rarely perforation of the esophagus or stomach may be the result of marked ulceration in the contents of the hernia.

Conventional roentgenograms of the chest are not adequate in establishing the diagnosis of hiatal hernia. Large herniae may be depicted but confused with cysts, tumors, atelectasis and other lesions of the thorax and lower abdomen. A final and absolute diagnosis of hiatal hernia of any type can only be made by correlation of clinical and roentgenological findings of studies of the lower esophagus and upper stomach with contrast media. The prone and supine Trendelenburg positions must be employed during these studies and it is only fair and tremendously important to inform the roentgenologist that a hiatal hernia is suspected and ask him to find it. The finding of a hiatal hernia on roentgenologic examination does not necessarily establish that upper abdominal or lower thoracic complaints are the result of this lesion. Before recommending surgical intervention there must be, therefore, a complete survey of the cardiorespiratory, gastrointestinal and biliary systems.

The importance of examination of the esophagus and upper stomach endoscopically has probably not been emphasized sufficiently. According to Harrington[2], a reliable differential diagnosis can be made between the acquired short esophagus and congenital short esophagus by the appearance of the esophagogastric junction. In the latter type the esophagogastric junction is patulous and the endoscope will enter with ease. In the acquired type, resistance will be encountered.

Palmer[3] has recently reported the study of 82 adults with hiatus herniae in whom the diagnosis was made by repeated roentgenologic examinations in 78 per cent. The first examination, however, demonstrated the lesion in only 60 per cent of the cases. In the same series, examinations with the esophagoscope established the diagnosis in 86 per cent and in 80 per cent on the first examination. Gastroscopic studies revealed the hiatus herniae in 46 per cent and of these 39 per cent were detected on the first examination. These and other studies demonstrate the value of endoscopic examination. Moreover, occasionally one will find a carcinoma of the esophagus, near or at the gastroesophageal junction, which is the basic mechanical factor in the production of the hernia.

If an esophageal hiatus hernia is producing symptoms, surgical intervention should be employed. Too often these patients are treated conservatively, are able to carry on an uncomfortable life and reach an older age group before ulceration, hemorrhage or other serious complications makes surgical intervention necessary and sometimes an emergency. There is probably no other lesion producing disabling gastrointestinal symptoms which is treated with so much indifference and procrastination.

There are sharp differences of opinion concerning the technics of repair of hiatal herniae. The principles, however, are well established. In the sliding or short esophagus type of hiatus hernia the lesion is a real sliding hernia in that the stomach makes up one side of the hernia sac. This sac must be eliminated either by plication or excision and closure. The hernia must be reduced. This reduction can be facilitated by making an incision through the muscular portion of the diaphragm, a short distance from the hiatus, and the viscera reduced into the abdomen through this incision. The opening at the esophagogastric junction must be large enough so that the esophagus is not constricted and yet snug enough to prevent recurrence. This is accomplished by the approximation of the crura of the diaphragm posterior to the esophagus.

Herniae of the paraesophageal or parahiatal variety are usually easier to repair than the sliding hiatus type. This is the case even in the very large herniations of the stomach through the diaphragm. The sac is easy to identify and free and reduction of the stomach or other viscera beneath the diaphragm is not difficult unless the contents of the sac have become incarcerated. The diaphragm is closed in the same manner as other hiatal herniae and the other principles are the same as described for the sliding short esophagus type.

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DISCUSSION

Dr. I. Snapper:—The three very interesting papers we have heard this morning certainly contain most valuable information.

Dr. Puestow, in his very instructive paper, insists that the condition most of us call achalasia, could just as well be named cardiospasm. In his opinion terminology has never brought medicine any further. Personally I have always felt that medicine is suffering because we have no clear cut semantics, that is, no exact definitions of the meaning of the terms we use. The same designation may indicate a completely different process to two different clinicians. The term cardiospasm should only be used if the cardia is spastic. This is not, however, the case in

achalasia because here the Ewald tube passes the cardia without any difficulty. In this condition the sphincter of the esophagus does not relax at the moment the food must pass from esophagus to the stomach. Therefore, the term achalasia, that is lack of relaxation, seems to be the correct designation.

It also seems at least doubtful whether the dilatation of the esophagus, which is so characteristic for achalasia, is a secondary effect. In three and four-year old children with achalasia, dilatation of the esophagus, as Dr. Putney has shown, is already present and in these children the so-called spasm can hardly have lasted long enough to bring about this anomaly of the esophagus. Thus it seems advisable to conclude that this condition is not due to a cardiospasm but to an imbalance of the innervation of the esophagus and the cardia.

Since we do not know the cause of achalasia, the importance of psychologic traumas for the etiology of this condition has been emphasized. Before accepting psychologic upheavals as the most frequent cause of achalasia the following should be considered.

It is also not rare to hear in the history of a patient with carcinoma of the esophagus that the difficulty in swallowing started first after a mental shock. Other patients with carcinoma of the esophagus will date the beginning of their complaints from the hasty drinking of a cup of very hot beverage. Thus the fact that the first signs of achalasia were observed after a psychologic disturbance may be significant but it still does not completely prove that the etiology of achalasia is limited to these factors alone.

In many cases there exists a remarkable similarity between pains caused by angina pectoris and by diseases of the esophagus. Head zones, that is painful areas which represent the projection upon the surface of the body of the irritation of the autonomous system of a visceral organ, are the same, irrespective of whether the heart or the esophagus is affected. In Orientals coronary thrombosis is one of the rarest diseases but carcinoma of the esophagus is very frequent. Thus, in China a European who is suffering from coronary thrombosis is usually diagnosed by the Chinese admitting physician as a patient with carcinoma of the esophagus. In the presence of crushing pain in the chest radiating to the left shoulder and arm, the Chinese resident who has never seen a coronary thrombosis, only considers the possibility of a serious disease of the esophagus. There is no doubt that large herniae of the esophagus can give rise to a syndrome simulating angina pectoris or coronary thrombosis. On the other hand I've seen many patients with typical signs and symptoms of coronary insufficiency but without any electrocardiographic changes in whom a small hiatus hernia was present. In such cases the decision as to whether operation of the small hiatus hernia is indicated to relieve the pains is often discussed with some heat. All the patients I have seen with this syndrome, however, died, operated or not, within a few years from an attack of coronary thrombosis. Therefore, although I accept the possibility that a large hiatus hernia can simulate a typical anginal syndrome, I am

disinclined to consider a small hiatus hernia as the cause of coronary-like pains. In patients with characteristic signs of coronary insufficiency and a small hiatus hernia, coronary sclerosis should be considered the cause of the pains even in the absence of electrocardiographic changes.

The thoracic surgeons however, do believe that a small hiatal hernia can cause a typical anginal syndrome. This explains why a few thoracic surgeons have approached me with the request for a prescription of a spasmolytic drug for their "attack of cardiospasm", when they actually suffered from coronary thrombosis.

The differentiation between gallbladder disease and pain attacks due to hiatal hernia is often difficult. In this connection the following rule is of importance. Any organ which is situated below the diaphragm cannot cause pain radiating to the neck or jaw. As soon as angina-like pains do radiate to the neck or jaw, the diseased organ must be situated above the diaphragm, usually the heart, occasionally a large hiatal hernia, but not the gallbladder.

Many patients with hiatal hernia have anemia. In every case of unexplained anemia, with or without pains in the chest, the presence of a hiatal hernia has to be considered. Hiatal hernia can easily cause hematemasis or melena. In some of these anemic patients with hiatal hernia—it may be difficult to find occult blood in the stool.

The medical treatment of achalasia and hiatal herniae is highly unsatisfactory. It is fortunate that now we have antibiotics. These drugs render the diagnosis of diseases of the esophagus by esophagoscopy and the treatment of achalasia by rapid dilatation less dangerous. Both esophagoscopy and forceful dilation occasionally cause rupture of the esophagus which in olden times required extensive operations to save the life of the patients. Now with streptomycin and other antibiotics these complications can easily be handled.

Some of the patients with achalasia and hiatal hernia are afraid of drastic dilatation and of surgical procedures. If in such patients the modern cholinergic drugs have failed to relieve the difficulties in swallowing an old medicament is sometimes helpful.

Twenty drops of a solution of 6 c.c. benzylbenzoate dissolved in 24 c.c. alcohol 70 per cent, given in milk before meals often facilitates the swallowing of these patients. Benzylbenzoate does not repair hiatal herniae, it will also not cure achalasia; nevertheless it may give at least temporary relief to these sufferers.

Dr. Owen H. Wangensteen:—At least one situation has been identified as being common to most instances of achalasia, and that is disappearance of the parasympathetic ganglion cells of Auerbach's plexus as occurs also in the congenital megacolon of Hirschsprung's disease. What causes the atrophy of these cells, of course, is not known nor can the disappearance of these cells be produced

experimentally. Hirschsprung's disease of the colon and rectum usually starts early in life; Finney of Baltimore (Surg., Gynec. & Obst. 6:624, 1908) described the condition in a newborn. Finney believed that lymphangiectasis with resultant fibrosis of the gut wall was responsible. In any case, Hirschsprung's disease of the colon is frequent enough in infants to justify the belief that it is an embryonic defect. On the other hand, achalasia of the esophagus ordinarily manifests itself later in life, rarely before puberty. Hirschsprung's megacolon and idiopathic megaesophagus have one thing in common: atrophy of the ganglion cells in Auerbach's plexus.

Esophageal dystonia is a term which has been employed by me in describing megaesophagus. Hypertonus of the terminal esophageal segment is present and atrophy of the more proximal portions. Unusual dilatation is a condition which distinguishes achalasia from other esophogeal obstructions. In an esophagus of normal tonus, obstruction does not cause great dilatation, only modest enlargement. Great dilatation appears to be a common denominator of long standing cases of esophageal achalasia. It was a common finding in the patients upon whom I resected the terminal esophagus and the acid-secreting area of the stomach, for megaesophagus (Ann. Surg. 134:301, 1951), Dr. Fred Cross, now of Cleveland, described absence of the parasympathetic cells of Auerbach's plexus as a common factor in all the cases in which I did the operation mentioned above for cardiospasm (Surgery 31:647, 1952).

Dr. Putney said that in the Philadelphia area they have not had good fortune with excision of the terminal esophagus and acid-secreting area of the stomach for cardiospasm. In the first case I did, I excised only the upper fourth of the stomach. That is not enough; I found it necessary to redo it, excising the entire acid secreting area. One thing that has been learned about the esophagus in recent years is its great sensitivity to injury by acid-peptic juice.

If anyone in Philadelphia or elsewhere removes only the hypertonic esophageal segment together with a fragment of the upper stomach, he is creating an esophageal cripple. With the esophageal sphincter gone, the acid-peptic juice from the stomach gains free access to the sensitive esophageal mucosa and hemorrhage ensues. Inasmuch as the vagi nerves are divided, a Heineke-Mikulicz pyloroplasty needs to be done to insure satisfactory emptying of the stomach. The Wendel and Heyrovsky-Gröndahl operations for megaesophagus, once so popular in this country, are now all passé because in correcting the esophageal obstruction, they invite esophagitis and hemorrhage.

Dr. Puestow's instrument for dilating esophageal strictures would appear to be a very useful apparatus. It would appear to circumvent the necessity of having the patient swallow a string for dilatation. I hope Dr. Puestow will get the manufacturers to make this instrument available so that every clinic will not have to go through the pains of labor of manufacture.

With reference to the statements of Dr. Putney and Dr. Puestow that the esophagus remains permanently dilated following hydrostatic dilatation for megaesophagus, I hope to be able to show that if an adequate myotomy of the esophagogastric juncture is done, dilatation disappears. In fact, I have come to feel that this technic of myotomy (Trans. Am. Acad. Ophthal. and Otol. 1953, p. 586) for achalasia is so simple and so effective that I have not in recent years done the more aggressive operation of excision of the acid-secreting area of the stomach together with the hypertonic-esophageal segment.

Dilatation with a bougie obviously gives only transient, if any continuing relief. Sifers and Crile (Gastroenterology 16:466, 1950) report clinical improvement in only 30 per cent of patients undergoing hydrostatic dilatation. A Mayo Clinic group of observers (Olsen, et al J. Thoracic Surg. 22:164, 1951) are more enthusiastic over the results of conservative hydrostatic dilatation for megaesophagus. All, who employ this latter method, admit that the esophagus remains dilated, even when the patient is symptomatically relieved. All who perform hydrostatic dilatation for megaesophagus admit that perforation is not only a possibility, but actually occurs more than occasionally.

Inasmuch as the Heller myotomy performed through a gastrotomy incision with a balloon distended with air on the end of a catheter relieves the condition as well as the dilatation of the esophagus—and without the same risk of blind rupture of the esophagus—I think that this method of management of megaesophagus presently will find increasing favor amongst patients who have the malady of cardiospasm.

Congenitally short esophagus is a condition diagnosed by roentgenologists more often than it actually exists. In a recent patient of mine, that diagnosis together with hiatal hernia was made by a competent roentgenologist. Neither condition was observed at operation done through an extrapleural sternal splitting incision which afforded an excellent view of the attic of the patient's abdomen. The stomach was completely within the distal thoracic abdomen; there was no hiatal hernia. Berg of Berlin described an ampulla in the esophagus as an occasional variant of the normal. This was, undoubtedly, what this patient had. Moreover, his esophageal stricture was probably owing to the regurgitation of acid-peptic juice. A tubular gastric resection accompanied by a Heineke-Mikulicz pyloroplasty was done. The patient recovered completely and has not needed additional dilatations. I have seen other such cases. Dr. Blades favors repairing hiatal herniae from within the thorax. The movie which Dr. Blades showed suggests that this is not a simple operation done from within the thorax. Harrington of Rochester, Minnesota, who has seen Allison of Leeds (Surg. Gynec. & Obst. 92: 419, 1951) do the operation from within the thorax, tells me that Allison does it essentially as a combined thoracoabdominal procedure cutting widely through the diaphragm. A few years ago, I too repaired hiatal hernia through the thorax. In order to insure a satisfactory repair, however, it was found necessary to cut

into the left leaf of the diaphragm, migrating the esophagus to the left (Ann. Surg. 129:185, 1949). When the extrapleural sternal splitting incision came into vogue in this clinic, simple apposition of the crura about the subdiaphragmatic esophagus by suture came to supplant all other methods. It is simple; moreover, it is very effective. One of my associates, who had spent a portion of his training in a hospital where the Allison operation for hiatal hernia was standard practice, remarked one day that the exposure provided by the sternal splitting incision removed all the mystery from the operative correction. In fact, the extrapleural sternal splitting incision makes repair of hiatal hernia by sutural apposition of the esophageal crura as simple as the repair of an indirect inguinal hernia.

Dr. F. J. Putney:—I am glad to have been able to hear Dr. Wangensteen's views. He and I differ on several things, one or two of which I should like to mention. After excision of the lower end of the esophagus and stomach, his group found an absence of Auerbach's plexus in the lower esophagus. We examined a number of these specimens and our pathologist found that Auerbach's nerve fibers were present in all of the excised specimens.

In regard to the dilatation, I should like to mention that many men are using mercury bougies in achalasia. These are not satisfactory, as everyone here has brought out, because of the fact that they do not stretch the hiatus. Actual avulsion has to be done.

I agree with Dr. Wangensteen that after the lower end of the esophagus becomes dilated, it remains permanently dilated and does not return to normal by dilatation, but the patients are clinically improved and gain weight. They have to be dilated once or twice, sometimes as many as six times and, after that, they usually require no extended treatment. The same thing is accomplished, one by surgical means, and the other by mechanical means.

Dr. Karver L. Puestow:—I have been struggling with this particular problem in attempting to determine what the mechanism of it is, whether that of achalasia or cardiospasm. I have been so frustrated that I have abandoned any attempt to figure out how these patients get that way. It does not make any difference to me whether it is called achalasia or cardiospasm, so long as we know what we are talking about. The names apply only to theoretical considerations as to the nature of the mechanism.

This whole subject seems to me to have been handled adequately both in the more radical approach to the problem, and by means of conservative measures. All I was attempting to do was to show that in most instances in which conservative measures are available, there are simplified ways in which they can be handled. All one is attempting to do is to retain the continuity of the gastrointestinal tract.

In one of the cases I showed to you, which I did not have time to discuss completely, the patient had had a jejunostomy as a preliminary procedure, and

in that particular instance we were able to demonstrate that we could maintain the continuity of the gastrointestinal tract, and after all, from the standpoint of the patient that is most important.

In these problems the surgeon and the internist, the esophagoscopist and the gastroenterologist, all have to work hand in hand. Some of these patients who may be treated conservatively in the beginning, eventually come to surgery, and some cases which are treated surgically may soon after require esophageal dilatation and be very, very comfortable thereafter.

I think that brings out one point. What we are looking for, after all, is a good functional response, and when I mentioned the fact that we should study our patients and not the x-rays, I was attempting to point out that too frequently progress studies are made by means of the x-ray, and further treatment is suggested by x-ray examination and not by means of the clinical progress of the patient.

Just as the case which starts out with conservative measures may eventually go to the surgeon, so some patients who have gone to the surgeons first quickly return for postoperative dilatation. If you will, visualize in the first instance a case of uncomplicated cardiospasm. In the beginning there is a short, tapered area of occlusion, which may eventually become elongated as the result of esophagitis. This is what I referred to as an organic change following a functional disturbance. In these instances one probably better send the patient to the surgeon and not attempt dilatation, because with the development of secondary cicatrix stenosis these patients do better if treated surgically.

On the other hand, when the surgeon does a gastrectomy and the patient has, as a result, an end-to-side anastomosis of the esophagus to the jejunum, one of his problems is the development of stenosis at the suture line. In passing a guide in such a situation it does not make any difference whether the guide tip extends into the upper or into the lower loop, it is only necessary that a sufficient length of guide wire be passed to enable dilatation of the stoma. The tissue at the stomal site is tough and does not rupture easily. If the surgeon, therefore, has made a good anastomosis, the stoma is much more resistant than the normal esophagus above, so that one may use considerable pressure in dilating through such an area without danger of rupture. Unfortunately, we do not have the opportunity of following these cases for a very long period of time.

I don't know whether Dr. Wangensteen agrees with me, but it seems to me that the surgeon is in a state of ectasy because of the fact that he is able to bring the stomach up, maintain its nourishment and establish an anastomosis at any level he desires. But the end results with the patients who have carcinoma are not very good. The mortality rate is extremely high both operatively and immediately postoperatively. One does not have an opportunity to follow these cases for a very long period of time.

I would be very happy if Dr. Wangensteen has any comment to make about that.

Dr. Brian Blades:—I have neglected to mention the importance of posture in patients with hiatal herniae. Their pain is usually relieved by the erect posture and before treatment if one observes these patients, they sit at the dining room table like a West Pointer, just as straight as possible with no sag. The posture relationship is one of the most important points in differential diagnosis, particularly in cases where coronary type of pain is present.

I am happy that the exercise tolerance test was mentioned as a differential point in distinguishing coronary pain from the pain of hiatal hernia.

Regarding the size of the hernia and the symptoms which may be produced, there is no true or sharp correlation between the size of the lesion and the various symptoms.

Dr. Wangensteen has asked me to comment on the crura. It is very difficult to locate accurately the crura by palpation from above. One must open the diaphragm and palpate from below to really distinguish these important structures.

HORMONAL INFLUENCES UPON THE STOMACH*

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Peptic ulcer has often been designated as a stress disease and the significance of chronic emotional and physical duress in its pathogenesis has been recognized for many years. The mechanism, however, by which chronic stress and alarm are transmitted to the stomach has been considered to be primarily by way of the vagus nerve.

The purpose of this report is to present evidence that emotional and physical stress is also mediated to the stomach by a purely hormonal pathway initiated in the cerebral cortex and transmitted to the stomach by way of the hypothalamus, pituitary and adrenal glands. This mechanism is independent of the vagus nerve and the gastric antrum.

A number of systemic stress factors (such as anoxia, shock, pain, infections, burns, trauma, etc.) or physical stimuli (muscular exertion, fatigue, temperature changes) as well as chronic emotional stress (rage, fear, anxiety, frustration) lead to the release of adrenocorticotrophic hormone by the pituitary probably under hypothalamic control. Adrenocorticotrophic hormone in turn activates the adrenal cortex to release a number of steroid hormones including cortisone and cortisone like compounds (Compound B, E, and F).

These adrenal steroids stimulate the gastric glands to secrete a considerable increase in acid and pepsin and may induce severe epigastric pain, cause reactivation, perforation or hemorrhage of a pre-existing peptic ulcer or produce a peptic ulcer de novo (Fig. 1)[1-5].

The repeated administration of adrenocorticotrophic hormone to patients over a period of time simulates chronic stress and elicits many of the same responses as those observed after chronic physical, systemic or emotional duress, namely a decrease in the circulating eosinophiles in the blood and an increase in the urinary excretion of 17-ketosteroids and 11-17 oxysteroids.

In the studies to be described the gastric secretory response to stress and hormone stimulation was evaluated by measuring the basal secretion of acid and pepsin in the gastric juice obtained by continuous aspiration for one hour and by 12-hour nocturnal aspiration. The peptic activity of the stomach was also evaluated by measuring the 24-hour excretion of pepsinogen in the urine (uropepsin).

RELATIONSHIP OF UROPEPSIN TO GASTRIC PEPSIN

The uropepsin excretion in the urine reflects the peptic activity of the stomach. It is derived from the secretion of pepsinogen directly into the blood stream by the peptic cells. Pepsinogen is then transported to the kidneys and is excreted in the urine as uropepsin. Uropepsin has been shown to be an accurate index of gastric juice pepsin and represents a constant fraction of the gastric secretion, approximating one per cent. With the administration of adreno-

```
Alarm and Stress
      ↓
Hypothalamus                          Parietel Cell  - - ➤  HCl
      ↓                                      ↑
Anterior Pituitary                          /
      ↓                                     /
     ACTH - - ➤ Adrenal Gland - - ➤ Adrenal - - ➤ Peptic Cell - - ➤ Gastric Pepsin
                                    Corticoids              ↓
                                                     Blood Pepsinogen
                                                            ↓
                                                     Urinary Uropepsin
```
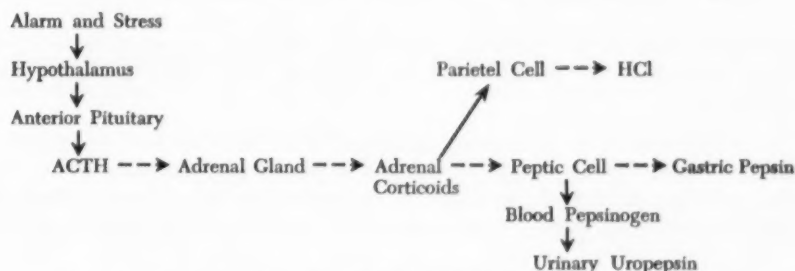
Fig. 1—Hormonal pathway by which stress is mediated to the stomach.

corticotrophic hormone or cortisone the increase in urinary uropepsin closely parallels the increase in gastric juice pepsin, and the exocrine-endocrine ratio remains constant (Tables I and II)[6,7].

EFFECT OF ADRENAL STEROIDS UPON
GASTRIC SECRETION AND UROPEPSIN EXCRETION

The administration of adrenocorticotrophic hormone, cortisone, Compound F or Compound B for a period of days or weeks induces a significant increase in the basal and nocturnal gastric secretion of acid and pepsin approximating 200 per cent with a similar concomitant increase in uropepsin excretion. The levels attained during hormone stimulation equalled or exceeded those observed in patients with active duodenal ulcer (Table III). These observations have been verified by others in both humans and animals[8].

The mean normal uropepsin excretion of 100 patients with no demonstrable gastric disease was 2,350 per 24 hours, while 215 patients with active duodenal

or gastric ulcer excreted a mean of 6,775 units per 24 hours, representing an increase in uropepsin excretion of 188 per cent (Table IV).

Patients experiencing epigastric distress during hormone therapy or developing a peptic ulcer, perforation or homorrhage demonstrate a coincident peak uropepsin excretion and an increased gastric acid and pepsin secretion invariably in the ulcer range.

With acute physical stress such as that induced by severe pain, anoxia, burns, trauma, fractures, surgical procedures, etc., the urinary excretion of

TABLE I
AVERAGE INCREASE IN PEPSIN AND UROPEPSIN DURING ACTH STIMULATION

Gastric pepsin (units per c.c.) 130%
Gastric pepsin (units per hour) 169%
Urinary uropepsin (units/24 hrs.) 194%

uropepsin increases to exceedingly high levels, with peak values averaging 22,000 units, exceeding the levels ordinarily observed with active duodenal ulcer (Table IV).

Under conditions of chronic emotional stress such as fear or anxiety or chronic physical stress such as that produced by dehydration, pain, fever, burns and radiation therapy the increase in uropepsin excretion is less marked than that observed with acute physical stress. The range in this group is from 7,000

TABLE II
ENDOCRINE-EXOCRINE PARTITION OF PEPSINOGEN

Control	ACTH	Recovery
1.2%	0.3%	0.9%
0.3%	0.3%	0.3%
Vagotomy 1.1%	1.1%	1.0%

to 18,000 units with a mean of 10,000 units, again exceeding the ulcer levels (Table IV).

The effect of chronic emotional stress produced during a Ph.D. examination is demonstrated by a 26-year old student whose uropepsin excretion increased from control levels of 2,359 units prior to the examination to 7,600 units during the examination period of 14 days. This increase in uropepsin excretion was associated with moderately severe epigastric pain both day and night. Several weeks after the examinations were completed the symptoms disappeared and the uropepsin excretion returned to normal levels.

The mechanism by which acute and chronic physical and emotional stress produce an increase in uropepsin excretion is probably by adrenal cortical stimulation, since a concomitant fall in the circulating eosinophiles and an increase in the ketosteroids in the urine can be demonstrated in each case.

TABLE III

AVERAGE INCREASE IN GASTRIC ACID, PEPSIN, AND URINARY UROPEPSIN DURING ACTH ADMINISTRATION IN NORMALS AND AFTER VAGOTOMY

	Normal	Vagotomy
Gastric pepsin (units/hour)	186%	160%
Uropepsin (units/24 hours)	161%	281%
Free HCl (mg./hr.)	241%	205%

EFFECT OF ADRENAL STEROIDS AND STRESS UPON GASTRIC SECRETION AFTER VAGOTOMY

Adrenocorticotrophic hormone was administered to patients upon whom a transthoracic vagotomy had been performed for the treatment of duodenal ulcer.

TABLE IV

STRESS FACTORS IN UROPEPSIN EXCRETION

	No. of Patients	Uropepsin Excretion (Units/24 Hours) Mean
Normal	100	2,350
ACTH and Cortisone	80	12,536
Physical Stress		
Acute	25	22,000*
Chronic	20	10,000*
Active Duodenal and Gastric Ulcer	215	6,775
Vagotomy		
Basal Conditions	13	6,500
ACTH or Stress	6	24,000*
Resection of Gastric Antrum		
ACTH or Stress	12	26,000*
Vagotomy and Subtotal Resection		
Marginal Ulcer	5	9,045

*Denotes Maximum Response

These patients had been essentially asymptomatic following their vagus section, and x-rays revealed no evidence of ulcer activity. Insulin tests demonstrated no increased acid or pepsin responses indicating that the vagus nerve had been severed successfully.

Vagotomy does not alter the uropepsin excretion. A mean of 6,500 units was observed in the vagotomized patients, approximating the average level excreted by peptic ulcer patients without vagotomy (Table IV).

The acid, pepsin and uropepsin response to hormone stimulation or stress in the vagotomized patients did not differ from those whose vagi were intact. The administration of adrenocorticotrophic hormone to vagotomized patients produced an increase in acid and pepsin approximating 100-150 per cent and an increase in uropepsin excretion approximating 200 per cent (Table I-IV).

In gastric fistula dogs with bilateral vagotomy an increase in gastric acid, chloride and pepsin secretion with a decrease in gastric secretory sodium and potassium occur within four to five hours of adrenocorticotrophic hormone administration intramuscularly.

TABLE V

EFFECT OF STRESS UPON UROPEPSIN EXCRETION

	Uropepsin Excretion Units/24 Hours
Normal	1,500-2,500 units/24 hours
Surgery	14,000 units/24 hours
Radiation	(6,400 control) 26,248-39,000 (air)
Burns	6,125— 2 days 12,140— 4 days 14,720— 5 days 10,251— 7 days 4,940—14 days

Surgical procedures, in general, induce sufficient stress and adrenal activation to produce a fall in circulating eosinophiles and an increase in 11-17 oxysteroids, with a gradual return to preoperative levels in 4-5 days. The surgical vagectomy procedure itself produces large increases in uropepsin excretion demonstrating that the gastric gland can respond to surgical stress in the absence of the vagus nerve. A rise from preoperative levels of 5,000 units to 52,000 units may be seen on the third to fourth day following the vagectomy, with a return to preoperative levels in 5-7 days, paralleling the fall in the urinary corticoids. Vagotomized patients undergoing subsequent surgical procedures similarly demonstrate a considerable increase in uropepsin excretion (Table V).

EFFECT OF BANTHINE AND ATROPINE UPON THE GASTRIC RESPONSE TO ADRENOCORTICOTROPHIC HORMONE OR CORTISONE

Atropine in doses of 2.4 mg. daily and Banthine in dosage of 150-300 mg. daily did not alter the uropepsin excretion or the uropepsin response to adreno-

corticotrophic hormone or cortisone stimulation in normals or in duodenal ulcer patients.

EFFECT OF ADRENAL STEROIDS UPON GASTRIC SECRETION IN PATIENTS WITH RESECTION OF THE GASTRIC ANTRUM (SUBTOTAL GASTRECTOMY)

The stomach in the absence of the gastric antrum can respond to stress. Patients with subtotal gastrectomy respond to stressful and adrenocorticotrophic hormone stimulation in the same manner as patients whose antra or vagi are

TABLE VI

UROPEPSIN EXCRETION IN PITUITARY AND ADRENAL INSUFFICIENCY

	Patients	Uropepsin Units/24 Hours
Normal	90	2,300

Hypopituitarism
   Untreated
   ACTH replacement therapy
	5
	3
		505
		1,882

Addison's disease before cortisone
   therapy
   During cortisone replacement
	10
	10
		543
		2,960

intact if adequate gastric mucosa is present. The uropepsin levels of patients with subtotal resection during periods of stress either surgical, adrenocortical or emotional increase to ulcer levels ranging from 20,000 to 50,000 units.

Patients who have undergone both vagotomy and subtotal resection for chronic duodenal ulcer and who subsequently develop a marginal ulcer, present uropepsin levels averaging 9,045 units, demonstrating again that the gastric

TABLE VII

EFFECT OF ADRENAL AND PITUITARY HYPERACIDITY ON UROPEPSIN EXCRETION

	Patients	Uropepsin Units/24 Hours Mean
Normal	90	2,300
Adrenal and Pituitary Hyperactivity (Cushing's Disease)	10	5,500

gland may function at a stress level without vagal or antral influences (Table IV). The uropepsin response to stress of patients with combined vagotomy and antral resection appears unimpaired.

Effect of the Hypothalamus, Pituitary and Adrenal upon Gastric Resection

The influence of the hypothalamus on the stomach was demonstrated by inserting a radio coil (Harris coil) into the hypothalamus of dogs and producing chronic stimulation for three days. The uropepsin level of these dogs increased approximately 100 per cent returning to control levels after stimulation was discontinued. The hypothalamic stimulation produced an adrenal response as evidenced by a fall in the circulating eosinophiles in the blood.

The relationship of the pituitary and adrenal glands to the stomach is demonstrated by the marked diminution in uropepsin output in patients with hypopituitarism and Addison's disease. With glucocorticoid replacement therapy the values returned to normal. A definite increase in uropepsin excretion moreover is observed in patients with adrenal and pituitary hyperactivity (Cushing's disease) (Tables VI and VII).

Comment

The stomach may be considered as an endocrine-exocrine organ. The urinary level of uropepsin is a measure of the endocrine-like activity of the stomach resulting from the secretion of pepsinogen into the blood stream by the peptic gland. If the stomach is to be looked upon as an endocrine organ one would anticipate that it would be subject to outside endocrine influences.

The occurrence of gastrointestinal ulceration is an integral and prominent part of the alarm reaction in response to a number of stress factors such as nervous and emotional stress, temperature changes, muscular exertion and fatigue, infections, burns, anoxia, shock, and intracranial lesions. All of these alarming stimuli may be associated with an increased incidence of gastrointestinal ulceration, perforation, or hemorrhage and are accompanied by an increase in gastric acidity and pepsin. Chronic stress, fear, and anxiety, as well as hostility and resentment are similarly associated with an increased secretion of hydrochloric acid. The exact relationship, however, between the acute gastroduodenal erosion of the alarm reaction and chronic peptic ulcer in man has not yet been classified, but it is reasonable to postulate that chronic stress could produce chronic peptic ulcer.

Moreover, peptic ulceration, hemorrhage, or perforation following trauma, burns, shock, brain tumors, as well as the "air raid ulcer" may be on the basis of a hypothalamic-pituitary-adrenal gastric mechanism since increased adrenal activity can be demonstrated under all of these conditions. It should be emphasized that peptic ulcer is exceedingly rare in the presence of adrenal insufficiency where gastric acidity and pepsin are at a low level. If the Addisonian patient is treated with cortisone, then peptic ulcer may occur, again emphasizing the significance of the adrenal steroids in the pathogenesis of peptic ulcer.

The intimate relation between acute and chronic stress and adrenocortical hormone output has been thoroughly elaborated in recent years and there is considerable evidence that the many forms of stress, described above, and stress simulated by adrenocorticotrophic hormone are on a common hypothalamic-pituitary-adrenal basis.

Adrenocorticotrophic hormone does not act directly on the gastric gland but induces a secondary outpouring of adrenal cortical steroids which then stimulates gastric secretion. This is proven by the fact that adrenocorticotrophic hormone does not affect gastric secretion in Addisonian patients while adequate doses of cortisone or Compound F and B in this group produce the usual gastric acid, pepsin and uropepsin response. In hypopituitarism, however, where the adrenal is able to respond to stimulation, adrenocorticotrophic hormone produces the customary gastric response, again demonstrating the hormonal pathway to the stomach (Table VI).

Cortisone and Compounds F and B are the only steroid hormones affecting gastric secretion in our studies so far. No uropepsin response was demonstrable after prolonged administration of desoxycorticosterone, testicular and adrenal androgens, and potent estrogen preparations. The only pituitary hormone which induces a uropepsin response is adrenocorticotrophic hormone; other hormones such as growth hormone, thyrotropic stimulating hormone and gonadotropin are ineffective. From the above evidence it would appear fairly definite that adrenocorticotrophic hormone, by activating the adrenal cortex and releasing adrenal glucocorticoids, exerts a specific influence upon gastric secretion. No other steroids or pituitary hormones tested have influenced gastric secretion to date.

SUMMARY

Although the gastric response to stress is undoubtedly mediated through the vagus nerve, the evidence presented strongly suggests that an additional hormonal mechanism is involved which is intimately associated with the General Adaptation Syndrome.

Chronic emotional and physical stress stimulates the stomach to secrete increased acid and pepsin by way of a humoral mechanism involving the hypothalamic-pituitary-adrenal-gastric axis independent of the vagus nerve or the gastric antrum. There appear to be significant hormonal influences in addition to neural stimuli in the pathogenesis of peptic ulcer. Potentiation of the hormonal pathway may explain the mechanism by which ulcers may recur in patients whose vagus nerves have been satisfactorily severed. Other factors in the pathogenesis of ulcer such as gastric motility, tissue resistance and vascular influences remain to be determined.

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EXPERIMENTAL METHODS FOR THE EVALUATION OF THERAPEUTIC AGENTS IN PEPTIC ULCER[*]

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The physician who treats patients with peptic ulcer is frequently urged to employ new therapeutic agents or procedures. Often, in the support of claims for the advantages of these innovations, results in the treatment of experimental ulcers in animals are presented. If the factors known to be concerned with the formation of the particular ulcer are not recalled and if comparable data with other therapeutic agents are not available, the significance of the results may be doubtful. The material to be presented was collected from the literature in an attempt to provide a background for interpreting the significance of beneficial effects in the treatment of experimental peptic ulcers.

The pylorus-ligated rat has been widely used as an assay for therapeutic agents in the treatment of ulcer. As Shay et al[1] reported, this procedure is attended

TABLE I

EFFECT OF OPERATIONS ON ULCERATION IN THE PYLORUS-LIGATED RAT

Type of Operation	Number of Rats	Percentage Protected	Author
Bilateral Vagotomy	29	100	Madden et al[2]
Ligation of the Ureters	25	96	"
Ligation of Bile Duct	22	100	"
Bilateral Adrenalectomy	18	89	Haroutunian et al[5]
Bilateral Nephrectomy	17	65	"

by the formation of ulcers in almost all animals operated upon. At least two factors are of importance in the genesis of this ulcer. Madden, Ramsburg, and Hundley[2,3], as well as Haroutunian et al[4,5] have been able to correlate the effectiveness of procedures or agents in preventing ulceration with their depressing effect upon gastric secretion. Donald and Code[6] have shown that draining the stomach through cannulae after pyloric ligation also prevents ulceration, although no change occurs in gastric secretion. The presence of obstruction, gastric retention, and the attendant effect upon the gastric mucosa differentiates this type of experimental ulcer from the others to be discussed.

The results of various operations performed on the pylorus-ligated rat are shown in Table I. Bilateral vagotomy gave 100 per cent protection against ulceration. This was associated with a reduction in gastric secretion. Bilateral adrenalectomy gave 89 per cent protection. This is of interest in view of current concepts of the effect of the adrenal glands upon gastric secretion. Ligation of the ureters might cause retention of an antiulcer factor in the urine, while ligation of the bile duct might exclude significant factors in the bile, but the 65 per cent protection afforded by bilateral nephrectomy suggests a nonspecific effect possibly operating through a decrease in gastric secretion[2,5,6].

The effectiveness of various drugs in reducing ulceration in this experimental ulcer is shown in Table II. The lowered incidence of ulceration is reflected by the "ulcer index" which is computed by multiplying the percentage of animals with ulcers by number of ulcers graded 1-5 or 1-6. Various anticholinergic drugs were partially effective in reducing the ulcer index, although in no instance as effec-

TABLE II

EFFECT OF DRUGS ON ULCERATION IN THE PYLORUS-LIGATED RAT

Drug	Number of Rats	Ulcer Index	Author
Controls	345	3.45	Barrett et al[7]
Atropine (8.0 mg./Kg)	10	0.3	"
Banthine (1.3 mg./Kg)	12	1.27	"
Antrenyl (500 mg./Kg)	12	0	"
"Enterogastrone" i.p.	11	0.5	Haroutunian et al[4]
Oil of Turpentine & Peanut Oil i.p.	8	0.06	"

tively as vagotomy[4,7]. Atropine, Antrenyl, and Banthine all showed significant degrees of protection.

"Enterogastrone" intravenously was inneffective, but given intraperitoneally, it exerted a protective effect[4]. Since intraperitoneal turpentine and peanut oil was equally effective, the authors concluded that the effect was a nonspecific one and paralleled the degree of toxicity and the depressing effect upon gastric secretion. Similar results were obtained with active and nonactive urine extracts. It would seem unjustified to use the pylorus-ligated rat as an assay for antiulcer substances which exert their effect on local resistance rather than through a depression of gastric secretion.

The gastric and duodenal ulcerations induced by the daily injection of histamine-in-beeswax have also been extensively studied. In their original article, Hay et al[8] reported that 12 of 13 dogs treated in this fashion developed ulcers: 8 in the duodenum and 4 in the stomach. In guinea pigs, the incidence of ulceration was

less: 6 of 10 animals developed ulcers. In view of the well known effectiveness of histamine in stimulating the parietal cells to produce hydrochloric acid and the relatively small increase in the secretion of pepsin, this experimental ulcer appears to be the result of prolonged hyperchlorhydria. In acute experiments in guinea pigs, however, Williams[9] has shown that in the early stages of development of the ulcer, (within 4-6 hours after the administration of histamine) the gross and microscopic character of the erosions suggest an ischemic origin, and Brun[10] has found that guinea pigs developed gastric ulcers after intraperitoneal histamine in the presence of gastric secretion with a pH of 4.5 or greater. Therefore it seems probable that local areas of diminished resistance play a role in some types of histamine-induced ulcers.

The effectiveness of various operative procedures in reducing the subsequent incidence of histamine-in-beeswax induced ulcers in dogs is shown in Table III[11,12].

TABLE III

EFFECT OF PREVIOUS OPERATIONS ON ULCERS INDUCED BY HISTAMINE IN DOGS

Type of Operation	Number of Dogs	Number of Dogs with Ulcers	Author
Controls	17	8	Lillehei et al[11]
50% Gastric Resection	4	1	"
Vagotomy	18	0	"
Vagotomy & 50% Resection	8	3	"
Vagotomy & Gastroenterostomy	6	3	"
Celiac Ganglionectomy	10	5	Holmes[12]

The need for controls is well illustrated since only 8 of 17 unoperated dogs developed ulcers. The addition of a drainage procedure appeared to lessen the protective value of vagotomy.

The effect of two therapeutic agents which have had trials in the management of human peptic ulcer are shown in Table IV. Neither "enterogastrone" nor Benadryl exhibited a protective action against the histamine induced ulcer[13,14].

A number of interesting potentiating factors in the production of histamine-in-beeswax induced ulcers in dogs have been noted. Thus nicotine[15] and muscular fatigue[16] increased the incidence of ulceration. Both of these factors have been thought to be significant in human peptic ulcer. It is interesting that muscular fatigue decreased gastric secretion in these animals.

Perhaps the best known among experimental technics for producing peptic ulcers is that described by Mann and Williamson[17] in which the stomach and duodenum of a dog are divided and the stumps inverted. The jejunum is transected

in its midportion and the proximal portion anastomosed to the distal ileum, while the distal portion is joined to the stomach. This shunts the duodenal, hepatic, and pancreatic secretions into the ileum and exposes the jejunum to the direct action of gastric secretion. In their original report, Mann and Williamson reported that 14 out of 16 animals operated upon developed ulcers in the jejunum at the site of the anastomosis. Although careful attention must be given to the details of handling these animals, the more important factors in the genesis of the ulcer

TABLE IV

EFFECT OF DRUGS ON ULCERS INDUCED BY HISTAMINE IN DOGS

Drug	Number of Dogs	Number of Dogs with Ulcers	Author
Controls	10	6	Grossman et al[13]
"Enterogastrone"	10	8	"
Controls	6	4	Friesen et al[14]
Benadryl	7	6	"

have been thought to be the state of nutrition, gastric hyperacidity, and diminished local resistance of the jejunal mucosa.

Singer, Sporn, and Necheles[18] reported recently an analysis of digestion and absorption in Mann-Williamson dogs. They found no significant change in glucose tolerance, chylomicron counts, or free fat in the stool. There was a flattening of the blood amino acid nitrogen curve after the administration of gelatin but not after glycine. This was interpreted as evidence of impaired protein digestion.

TABLE V

EFFECT OF OPERATIONS ON ULCERATION IN THE MANN-WILLIAMSON DOG

Type of Operation	Number of Dogs	Number of Dogs with Ulcers	Survival of Dogs with Ulcers	Author
Controls	9	9	68 days	Storer et al[20]
Vagotomy	11	5	73	"
Antrum Resection	6	2	168	"
Vagotomy & Antrum Resection	6	1	153	"

The effect of this operation upon gastric secretion has been debated but the recent report of Storer et al[19] showed that Mann-Williamson dogs equipped with Heidenhain or denervated pouches increased their gastric secretion 100 per cent in terms of milliequivalents of hydrochloric acid per 24 hours as compared with preoperative values. They interpreted these results as evidence that the hypersec-

retion of the Mann-Williamson dog was mediated through hormonal factors of the gastric antrum. They also showed that the incidence of ulceration was reduced by the intragastric drainage of duodenal content.

The results of various operations on Mann-Williamson dogs are shown in Table V. Storer et al[20] have found that although vagotomy has some protective value against ulceration, antral resection appears to have the greater beneficial effect. The combination of antral resection and vagotomy gave the highest number of dogs free of ulcer and provides some evidence in support of the current interest in a limited gastric resection and vagotomy in the treatment of human peptic ulcer.

Various drugs have been employed in the treatment of this experimental ulcer and Table VI shows some of the results. Some antacids[21,22], anticholinergic

TABLE VI

EFFECT OF DRUGS ON ULCERATION IN THE MANN-WILLIAMSON DOG

Drug	Number of Dogs	Number with Ulcers	Survival of Dogs with Ulcers	Author
Controls	20	19	56-280 days	Fauley[21,22]
Creamalin	23	23	56-245	"
Aluminum Phosphate	20	3	60-178	"
Controls	15	12	95	Harkins[23]
Banthine (5 mg./lb.)	22	7	57	"
Atropine (1 mg./4 lbs.)	5	1	82	"
"Enterogastrone" Subcut. 3 mos.	5	1	120	Ivy[24]
Controls	19	14	89	Sandweiss[25,26]
Uroanthelone (Kutrol)	10	2	196	"
Corticotropin	16	5	139	"
Cortisone	10	7	149	"

drugs[23], and antiulcer factors[24,25] have been found to reduce the incidence of ulceration. This experimental ulcer preparation has been particularly useful in demonstrating the action of the latter group of substance, including "enterogastrone" and urogastrone. Recently corticotropin and cortisone have been suspected of producing or activating human peptic ulcers. The results of Sandweiss et al[26] seem to show rather a beneficial effect of these hormones and call for further studies to explain this discrepancy.

One other type of experimental ulcer will be mentioned because it presents a type of ulcer which seems to be due entirely to hyperfunction of the gastric antrum. Dragstedt et al[27] have recently described a technic for producing ulcers which consists of removing the gastric antrum and transplanting it to the colon

as a diverticulum and performing a gastrojejunostomy. Five of six dogs operated upon in this fashion developed jejunal ulcers in 21-100 days. Hammer et al[28] have shown that transplanting the antrum in an antiperistaltic manner so that fecal material would tend to be excluded, reduces the incidence of ulceration. Therefore it appears that an ulcer due to hyperfunction of the gastric antrum which acts by means of gastrin to produce hyperchlorhydria in the remaining portion of the stomach, is available for the assay of therapeutic measures.

The four types of experimental ulcers discussed are not comparable to peptic ulcer in man with the possible exception of the jejunal ulcer after gastric resection or gastrojejunostomy. Procedures found to be of value in human peptic ulcer, however, have also, in general, been of protective value against experimental ulcers. The advantages of the pylorus-ligated rat are the high incidence of ulceration in controls and the relative ease in working with large numbers of animals. It has been used successfully as an assay for substances which reduce gastric secretion, but it is probably not satisfactory for studying agents which act primarily by increasing local resistance (antiulcer substances).

The histamine-in-beeswax induced ulcer in dogs has in common with human peptic ulcer the hyperchlorhydria and perhaps the diminished local resistance. Surgical procedures effective in man are effective in protecting against this ulcer. Because the incidence of ulceration in control animals is variable, studies showing advantages for newer therapies must be carefully scrutinized for adequate controls and a sufficient number of animals in order for the data to be significant.

The Mann-Williamson ulcer has proven a severe test of the value of therapeutic measures to be used in human peptic ulcer, but in general those measures proven of value in this experimental ulcer have also been useful in man. The factor of a hypersecretion mediated through the antrum may be the primary one in this type of ulcer. The value of antiulcer substances without effect upon gastric secretion has been demonstrated in this type of experimental ulcer.

SUMMARY

The four main methods of producing peptic ulcer in the experimental animal are:

1. Ligating the pylorus in the rat (Shay).

2. Histamine-in-beeswax in the dog (Hay).

3. Diverting the alkaline duodenal, biliary, and pancreatic secretions from the upper small intestine, while permitting gastric secretion to enter the jejunum (Mann and Williamson).

4. Transplanting the gastric antrum to the colon and producing a hypersecretion of hydrochloric acid from the remaining stomach which then enters the jejunum through a gastrojejunostomy (Dragstedt et al).

These animal preparations have been used by various investigators as a test of : 1. medical and 2. surgical measures which have been thought to be useful in the treatment of human peptic ulcer.

The data available are not extensive. In general, those measures found to be useful in the treatment of human peptic ulcer seem to have beneficial effects on the peptic ulcer produced experimentally in animals.

One or more of these animal preparations may be useful to assay and compare the efficacy of the various methods of treating peptic ulcer in man. Yet the final test is the effectiveness in man.

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THE PRESENT STATUS OF VAGOTOMY IN THE TREATMENT OF DUODENAL ULCER[*][†]

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The surgical treatment of duodenal ulcer has run through a gamut of procedures. Gastroenterostomy and pyloroplasty, which were at first designed for the mechanical relief of pyloric obstruction, were subsequently employed to cure the disease. While gastroenterostomy usually healed the duodenal ulcer, it gradually became evident that gastrojejunal ulceration with its severe pain, bleeding, and occasional perforation, developed in 16 per cent to 33 per cent of the cases. It was natural, therefore, that a more rational therapeutic approach developed when the physiology of gastric secretion was better understood, and the role of free hydrochloric acid in the genesis of ulcer was appreciated. This major attack was directed along lines which would abolish or lower the free hydrochloric acid. It was hoped this might be accomplished by either removing the cephalic or psychic phase, or the chemical phase of gastric secretion. It seemed logical to many that the section of the vagi which controlled the psychic phase of gastric secretion should be effective. Many technics for partial vagotomy were reported from 1920 to 1924, but these procedures were soon replaced by the operation, subtotal gastrectomy, which excised the pylorus and antrum, the areas primarily responsible for the chemical phase of gastric secretion.

This operation, especially the Billroth II type, has won increasing recognition in the United States as experience in technic was gained. The mortality, which in the beginning was high, has gradually fallen to about 1 per cent. By and large most patients who have had this operation are happy and satisfied with the results. Unfortunately, however, there are some who fail to gain weight, a few who suffer from the dumping syndrome, which is distressing, and 5 per cent who develop gastrojejunal ulceration. The test meals in this group of patients invariably reveal large quantities of free hydrochloric acid.

It has long been recognized that ulcers rarely develop in the presence of anacidity, for "no acid, no ulcer". In our series over 40 per cent of patients who have had a subtotal resection still have free hydrochloric acid. Theoretically they are all potential candidates for possible gastrojejunal ulceration. As early as 1929, Klein[1] conceived the idea of combining partial vagotomy and gastric resection in

order to eliminate the two important pathways of acid stimulation, thereby hoping to increase the incidence of achlorhydria. He published his experimental results in dogs of left vagus nerve resection and partial gastrectomy. In 1938[2], the follow-up results in 34 selected patients with subtotal gastrectomy, in which vagal fibers on the anterior wall of the stomach were divided, seemed to verify Klein's contention. In 1946 we began combining bilateral infradiaphragmatic vagotomy with resection, using it at first for patients in whom the preoperative acidity was high, and those in whom hemorrhage was a symptom, two categories prone to subsequent ulceration. It soon became apparent that the addition of vagotomy to gastric resection did not cause an increase in mortality although it did increase postoperative morbidity. A marked reduction in the free acid secretion, or an achlorhydria occurred in the majority of cases.

It might be interesting to review this series of patients with duodenal ulcer treated by subtotal gastrectomy from 1946 through 1952[3]. In about half of these, bilateral infradiaphragmatic vagotomy was added without undue difficulty. There was an increase in the incidence of postoperative pulmonary complications. Fol-

TABLE I

FOLLOW-UP RESULTS IN GASTROJEJUNAL ULCERS FOLLOWING GASTROENTEROSTOMY

Operation	Well		Recurrence		Total
	No.	Per cent	No.	Per cent	
Bilateral Vagotomy Alone	4	67	2	33	6
Subtotal Gastrectomy Plus Vagotomy	8	89	1	11	9
Subtotal Gastrectomy	15	75	5	25	20

low-up studies revealed that vomiting, abdominal distention, weight loss and the dumping syndrome, were not more frequent following the combined procedures than in those with resection alone. Diarrhea, however, was more prevalent, but in only one case was it disabling. The outstanding difference in the follow-up of those with subtotal gastrectomy and those with the combined procedure was in the incidence of gastrojejunal ulcer. There were 13 recurrences in 218 cases of resection alone (5.9 per cent), while to date in the combined group, there has only been one recurrence in 157 cases (6 per cent).

During the same period, 1946 to 1952, another group of 101 duodenal ulcer patients were followed[4]. These were treated by gastroenterostomy to which vagotomy was added in 73. It was felt that all these patients would not tolerate the magnitude of a gastric resection, and while that is our procedure of choice, we will not employ it on patients we believe to be poor surgical risks. This group includes those who are not chronologically, but physiologically 60 or over, those

who are obese, diabetic, hypertensive, or those with coronary artery insufficiency. It also embraces another group in which, because of massive infiltration surrounding the ulcer, the duodenal dissection might prove hazardous, and the closure extremely difficult, or impossible. In these categories of patients, a gastroenterostomy was performed, fully realizing its inadequacies, but appreciating that it entailed less risk. Whenever feasible, an infradiaphragmatic vagotomy was added. The mortality of these simple procedures in this group was 5 per cent, (verifying our preoperative fears as to risk), as compared to 0.7 per cent in the subtotal gastrectomy series. The incidence of gastrojejunal ulcer in the gastroenterostomy-vagotomy group was 10.3 per cent, and 15 per cent in those in whom gastroenterostomy alone was performed. It is realized that the number of cases in each group is too small to draw any conclusions which might be interpreted as significant. In other clinics[5] in which it has been a policy to perform gastroenterostomy and vagotomy as a procedure of choice, the results seem to indicate that the addition of vagotomy lessens the incidence of recurrent ulcer, but the original enthusiasm is not supported by a study of subsequent reports.

TABLE II

Follow-up Results in Gastrojejunal Ulcers Following Subtotal Gastrectomy

	Well		Recurrence		
Operation	No.	Per cent	No.	Per cent	Total
Vagotomy Alone	10	59	7	41	17
Additional Gastric Resection	9*	69	4	31	13

*Combined with Infradiaphragmatic Vagotomy in 4 Cases.

The addition of vagotomy to subtotal gastrectomy has not been adopted in the majority of clinics. The impression seems to be that the undesirable side-effects of vagotomy (which we have not observed too frequently), do not justify its use, and, moreover, that vagotomy should be reserved for that time when a gastrojejunal ulcer actually develops. We first employed vagotomy for gastrojejunal ulcer following an adequate subtotal gastrectomy, in 1939[6]. As we review our experience since then[7], it becomes evident that this simple procedure usually produces an immediate dramatic disappearance of the lesion, but it has not proven to be a panacea because too often gastrojejunal ulceration recurs (Tables I and II). We have learned that the best procedure in cases of gastrojejunal ulcer following gastroenterostomy is not a subsequent vagotomy alone, but rather a high subtotal gastrectomy combined with infradiaphragmatic vagotomy. Moreover one should never expect vagotomy alone for postgastrectomy ulcers to be effective if the previous gastrectomy alone has been inadequate, or if the stoma is obstructed. This type of case requires an additional resection, or correction of the mechanical difficulty.

There is also a group of subtotal gastrectomy cases, fortunately small, in which, regardless of the extent of the secondary gastric resection, even if combined with a complete vagotomy, unneutralized free acid is still secreted and ulceration recurs. In this type of case, a total gastrectomy is the only effective remedy and it has been performed reluctantly. We say "reluctantly" because most patients with total gastrectomy are not happy and present nutritional disturbances which require medical care throughout their life.

As we reflect upon our results of vagotomy combined with subtotal gastrectomy, we get an empiric clinical impression that this offers greater protection against gastrojejunal ulceration than when vagotomy is added subsequently to a subtotal gastrectomy, in which gastrojejunal ulceration has occurred. A statement such as this may be statistically unsound, especially when one considers the fact that the fundamental ulcer diathesis remains unchanged, and that recurrences have been seen 25 to 30 years after gastroenterostomy, and many years after subtotal gastrectomy. We can only state at present that our results seem to justify a continuation of subtotal gastrectomy combined with vagotomy because this procedure has neither produced an increase in operative morbidity, nor in the undesirable side-effects of vagus nerve division, and to date in our series it has definitely diminished the incidence of gastrojejunal ulceration.

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With Dr. Ferguson's permission, Dr. Weinstein will show some slides.

Dr. Weinstein:—(Slide) The following slides will summarize most briefly the ulcer material upon which our present concepts of the place of vagotomy in the treatment of duodenal ulcer are based.

During the period of seven years from 1946 to 1952, on the ward and private services of Dr. Colp at Mt. Sinai Hospital, we treated 546 cases of duodenal ulcer: 445 of these were selected for subtotal gastrectomy, chiefly because they were good risk cases. A gastroenterostomy was done for the remaining 101. This includes the poor risk cases plus a small number of relatively good risk patients on whom we wished to try the procedure of gastroenterostomy and vagotomy.

Vagotomy was added to 73 of the 101 patients who had gastroenterostomies, and in the large group of 445 gastrectomy cases, vagotomy was added to 207. In the poor risk gastroenterostomy patients, we had a 5 per cent mortality, a direct result of selection of cases, the poor risks being treated by the lesser procedure. In our good risk cases, treated by subtotal gastrectomy, our mortality was 0.7 per cent—three cases lost out of 445.

(Slide) Considering only cases treated by subtotal gastrectomy, we had 220 treated by subtotal gastrectomy alone, and 165 by the combined procedure. In reviewing the postoperative course in these two groups we noticed there was a somewhat longer average febrile period in the vagotomy group. We had more of the moderate severe reactions in the vagotomy group than in the gastrectomy alone. There were 2 mortalities listed in the vagotomy group, but we could not attribute them to vagotomy per se.

(Slide) In the review of the complications in general, the subtotal gastrectomy group, as compared to the gastrectomy and vagotomy group—the slide shows more pulmonary complications in the vagotomy-gastrectomy group. The rest of the complications were similar in the two groups.

(Slide) The overall follow-up results, from four months to over six years, shows that there was not any particular difference in the two groups, as far as vomiting was concerned. There were more cases with diarrhea in the vagotomy than in the other group. Severe pain occurred only in the gastrectomy patients. Bleeding, the result of gastrojejunal ulceration, occurred only in the gastrectomy group. Dumping was about the same in the two groups.

(Slide) An evaluation of the recurrence rate for the whole series. Our gastroenterostomy groups are not large enough to give us signficant figures. However I have listed them. We had a 15 per cent recurrence rate for gastroenterostomy, and 10.3 per cent for gastroenterostomy with added vagotomy. Following subtotal gastrectomy, we had 13 recurrences, or 5.9 per cent recurrence rate, and for subtotal gastrectomy with added vagotomy, we had only one recurrence, or 0.6 per cent. The difference between 0.6 and 5.9 per cent is statistically significant.

(Slide) The acidity studies are of some interest. When we review the three groups of cases, we find that gastroenterostomy and vagotomy does not give us nearly the percentage of achlorhydria tests as in the other groups, and the best results are obtained with subtotal gastrectomy and vagotomy, in which there is quite a high percentage of postoperative anacidity tests, following gruel, alcohol,

or histamine stimuli. In the night secretion tests, 98 per cent were antacid in the subtotal gastrectomy-vagotomy group.

(Slide) There are two slides in which I should like to give the results of vagotomy for gastrojejunal ulcer. In gastrojejunal ulcers treated by vagotomy alone, our results were poor, and that procedure was discontinued. We felt we should do a gastrectomy, and, whenever possible, add a vagotomy.

The best results were obtained with the group where vagotomy was added to gastrectomy, but the figures are not significant because the numbers are small.

(Slide) In the gastrojejunal ulcers which occurred following subtotal gastrectomy, we have a group of cases treated by vagotomy alone. There were 17 cases, 10 of which were well and 7 of which had recurrences, and at first we thought our results were going to be good with vagotomy alone, but we found a definite number recurred, so we had to do additional resection.

In summary, therefore, the present status of vagotomy in this group of patients is that of an ajunct procedure. It is added to a resection or gastroenterostomy, we think with some benefit.

DISCUSSION

Dr. I. Snapper:—The excellent paper of Dr. Gray is a convincing demonstration of the extensive changes of the secretion of acid and pepsin which take place under influence of corticosteroids. Dr. Gray's paper offers a solution of many problems which for many years seemed insoluble; for instance, why so many patients who suffered a coronary closure or a fracture developed a fresh ulceration or even a perforation of the stomach.

We now understand that these and many comparable developments are closely connected with the stress which causes secretion of corticosteroids and thereby increases the secretion of pepsin.

Important as these results are, Dr. Gray undoubtedly agrees that the formation of a peptic ulcer does not alone depend upon acid and pepsin production. Corticosteroids cause remarkable changes in many tissues, especially in the collagen all over the body. These collagen changes, perhaps even more than excessive acid and pepsin secretion, also may favor perforation of the stomach after the liberation of corticosteroids under influence of a stress phenomenon.

I am certain that Dr. Gray would not want us to forget the many other mysterious problems of the ulcer disease which develop in one individual but not in the next one although both have the same amount of acid and pepsin.

I am very much impressed by the results of the investigation of Dr. Brooks on the treatment of experimental ulcers in animals. The application of these results to patients will require great caution.

Among the native population of Indonesia peptic ulcer of the stomach was very infrequent whereas in North China this disease occurred frequently.

Since racial differences are of such importance for the development of peptic ulcer, the differences between ulcers of humans and animals must be immeasurable. This is also evident in the results Dr. Brooks has reported. It is well known that one rabbit can eat with impunity large quantities of belladonna leaves, sufficient to intoxicate a whole regiment of soldiers. Rats evidently also can stand terrific amounts of this drug, because in Dr. Brooks' experiments 8 mg. of atropine per kilogram of body weight were given to the rats.

The Mann-Williamson ulcers frequently develop in experimental animals within a week's or two weeks' time. If a patient has a complete derivation of the bile flow by a common duct drainage which may be kept open for one or more years, no tendency to ulcer formation develops.

Therefore, necessary and interesting as the study of experimental ulcers in animals is, application of these results to human pathology is fraught with dangers.

For eight years I have had the privilege to closely follow Dr. Colp's operations, and I have had ample opportunity to convince myself that his results in the surgical treatment of ulcer of the duodenum are outstanding. As a physician, of course, I am not in favor of mutilating operations. Nevertheless in many cases of peptic ulcers of stomach and duodenum, subtotal resection of the stomach cannot be avoided because the results of medical treatment of ulcers is so very poor, especially among the population of the hospital wards.

Dr. Colp has mentioned the study of the results of vagotomy by Dr. Klein. In this connection it is important that Dr. Talma, around 1890, reported ulcer formation under influence of electrical stimulation of the vagus nerve. Talma, who also devised omentopexy, was a professor of medicine in Utrecht, not a surgeon. He cut the vagus in the rabbit, stimulated the nerve with an electric current, and saw the development of peptic ulcers in the stomach of the rabbit. He explained the ulcer formation by a spasm of the muscularis mucosae under influence of vagus stimulation. This spasm closed off little nutrient arteries of the mucosa and the resulting ischemia was the cause of the ulcer.

This explanation may not be correct but the fact remains that the fundamental experiments on the influence of the vagus upon peptic ulceration of the stomach have been performed 60 years ago by Dr. Talma.

Dr. O. H. Wangensteen:—That the coordinators also learn in these programs is quite apparent. It would have been very difficult four or five years ago, when Dr. Snapper and I first began conducting these sessions, for Dr. Snapper to say publicly that the medical treatment for peptic ulcer was poor. Progress comes too in the minds of the coordinators.

It was a privilege to have had the opportunity of hearing the delightful paper by Dr. Gray; it points up an attack upon the problem of peptic ulcer which

needs to be pursued. I agree with Dr. Snapper that mere demonstration of increase in uropepsin does not per se denote increase of susceptibility to peptic ulcer. As Dr. Gray emphasized, under conditions of stress, uropepsin is increased and peptic ulceration may occur. A definite relationship between stress and ease of ulcer provocation has been demonstrated experimentally beyond question. This mode of attack upon the problem of peptic ulcer should prove helpful in bridging some of the defects in our knowledge.

While in attendance at this meeting, I have had the pleasure of seeing again my erstwhile colleague, Dr. Fred Mears, now of Santa Ana, California. A few years before World War II, Drs. Harry Hall, Lyle Hay, Fred Mears and I embarked upon the problem of ascertaining the effect of atropine upon the night secretion of patients with duodenal ulcer. We noted definite reduction in the volume and in the acidity in patients with duodenal ulcer having an indwelling duodenal tube—but not in the same degree reported subsequently by Dr. Lester Dragstedt and his associates (1943), attending vagotomy. Moreover, Hall, Hay and Mears found the decrease in gastric acidity to be greater than in patients who did not have peptic ulcer. These experiments were undertaken by us with a view to appraising the likely promise of bilateral vagotomy in duodenal ulcer. In fact, mention was made of our having performed vagotomy in the protocols of two patients operated upon for peptic ulcer in two papers published from this clinic (Surg., Gynec. & Obst., 70:58, 1940 and Ann. Surg., 112:626-670, October, 1940). Hall went to England late in 1940 and Hay went off with U. S. Hospital No. 26 in February, 1942. Mears continued the work and concluded from his observations that atropine was considerably more effective in the duodenal ulcer patients than in normals in the control of acid-peptic night secretion. We were not encouraged, however, to believe that these findings lent any support to the idea of control of acidity by abolishing vagus action. The observations of Mears (Surgery, 13:214-223, 1943) were published about the time that the paper of Dragstedt and Owens (Proc. Soc. Exper. Biol. & Med., 53:152, 1943) on vagotomy appeared.

That there is an ulcer problem is quite apparent. One only needs to look at the situation at the national level. The Vital Statistics of the United States indicates that an 85 per cent reduction in the mortality of appendicitis has occurred in the past 15 years; that in the past decade the mortality of bowel obstruction has been reduced by about 50 per cent. On the contrary, there appears to have been no decline in the mortality of peptic ulcer over the past 3 decades. I think if Dr. Snapper and other internists would say publicly more often that the medical treatment of peptic ulcer is ineffective, I think a great improvement in the mortality of peptic ulcer would come about in this country.

Dr. Snapper:—I said in the hospital population.

Dr. Wangensteen:—Dr. Snapper will have an oportunity to amend his statement.

Dr. Snapper:—No amendment to the statement. I said in the hospital population.

Dr. Wangensteen:—Dr. Snapper says he indicated mortality in the hospital population of patients with peptic ulcer. Some patients do not get to the hospital because they die of hemorrhage from their peptic ulcer before they can be hospitalized.

My colleague, Dr. James H. Casey (Minn. Med., **37**:448, 1954) studied the vital statistics of deaths from peptic ulcer in the State of Minnesota for 1952. There were 129 deaths from peptic ulcer in Minnesota in 1952; some of these died of hemorrhage before they got to the hospital. About 50 per cent of all peptic ulcer deaths were found to be due to hemorrhage, and in an additional number, some element of hemorrhage was also a factor. When we begin to face the problem of hemorrhage from peptic ulcer realistically, we will begin to reduce the mortality. And I would suggest that what happens in Minnesota probably occurs here in the District of Columbia as well as in your respective areas. Casey's study, I feel, should help point up the importance of hemorrhage in keeping the mortality of peptic ulcer higher than it should be.

Since 1930, deaths from peptic ulcer in this country per hundred thousand population have remained aproximately the same over this 25-year period, with a death rate of about 4.5 per 100,000 thousand population. The insurance companies do not fret too much over this, because 85 per cent of their death benefits are paid out to the survivors of patients who died of the four following ailments: heart disease, cancer, congenital anomalies and trauma. Diabetes and tuberculosis each account for approximately 3 per cent of the total death benefits paid out by insurance companies. Peptic ulcer deaths account for only one per cent of death benefits; appendicitis for 0.5 per cent. A great part of the wealth of this country is invested in insurance companies. Unfortunately it is not possible to dissociate money and disease completely.

In this discussion, I should like to speak primarily of my experience as a surgeon in dealing with the peptic ulcer problem. The stress factors which Dr. Gray spoke of are teaching us a great deal concerning the origin of peptic ulcer. I also enjoyed the presentation by Dr. Colp very much. Surgically, there seem to be many ways to Rome in dealing with the peptic ulcer problem. I am using some methods currently, which in the light of Dr. Colp's presentation, would be regarded as quite unconventional.

In 1951, when my good friend Dr. I. S. Ravdin of Philadelphia was Chairman of the Surgery Section of the A. M. A., he invited me to speak on segmental resection for peptic ulcer—an operation which Mikulicz described and abandoned in the year 1897. I accepted Ravdin's invitation to speak with great reluctance for I felt that the platform of the Surgery Section of the A. M. A. was not the proper spring-board from which to launch a subject which I felt would probably prove

quite controversial. A number of unkind things were said that day in Atlantic City about my efforts with segmental resection. My warm friend of many years Dr. Lester Dragstedt, for whom I have great admiration as well as affection, said that segmental resection for ulcer was doomed to failure and was a physiologically unsound operation. The basis for Dragstedt's criticism was that the antrum was retained in segmental gastric resection. I had been at pains in the presentation of the paper to point out that I felt there was an important difference between *retaining* the antrum and *excluding* it, as in the pyloric exclusion operation. And again, this was my defense in the rebuttal. In the intervening years, it has become quite clear that this difference which I emphasized then has since come to be appreciated as an extraordinarily important one. *Antral exclusion* invites recurrence; not so, if the antrum is left attached to the residual gastric pouch. Moreover, time has shown that segmental resection is a good operation. My associates and I have done more than 100 such cases. There has not been a single instance of peptic ulcer recurrence following this operation. In segmental resection as I have done it, we have found it necessary to add a Heineke-Mikulicz pyloroplasty because the pylorus is vagotomized in making a transverse division of the stomach. My colleague, Dr. Donald J. Ferguson, at our Veterans Hospital, finds that by taking pains to preserve the vagus nerve supply to the pylorus and antrum by preserving the gastrohepatic omentum intact, save over the area of resection it is not necessary to add the pylorotomy, unless the presence of obstruction or hemorrhage dictates the necessity of the performance of pyloroplasty.

Since the revival of segmental resection in our clinic 6 years ago, I have undertaken an additional operation, in the hope of finding a procedure which would thwart the ulcer diathesis and which would not be accompanied by the dumping syndrome. If it were not for some of the undesirable side-effects of the Billroth operations, whether done on the Billroth I or II plan, the search for additional operations, with which to combat the ulcer diathesis surgically, could be abandoned. These operations when accompanied by an adequate excision of acid-secreting area will prevent ulcer recurrence. The incidence of undesirable side-effects, however, has been too high—and so too with segmental gastric resection. With the avoidance of pylorotomy as Ferguson does segmental resection, the dumping syndrome will undoubtedly be less frequent. The operation which I have been doing is one which I called tubular resection a number of years ago (Surg., Gynec. & Obst., 70:59, 1940). I have more recently revived this operation in a new dress, adding a transverse gastroplasty, thus reconstructing a miniature gastric pouch. Moreover, inasmuch as the entire lesser curvature is not disturbed, the vagus nerve supply to the pylorus and antrum remains intact. A pyloroplasty, therefore, is added only in the presence of obstruction or when hemorrhage from a duodenal ulcer is present.

Some current studies in the laboratory on dogs with Heidenhain pouches suggest that tubular resection does not do away with the gastrin response to a meat meal in the dogs as do the Billroth operations and segmental resection.

The reason for this is not clear, but it would appear that preservation of the vagus nerve supply to the antrum may be responsible. I am proposing to do some tubular resections cutting the vagal twigs to the antrum in the gastrohepatic omentum to assess this difference in behavior.

You will note from the lantern slide review of unacceptable operations that I have listed the following: gastrojejunostomy, pyloroplasty, the small gastric resection (50 per cent or less), the Connell Fundusectomy, vagotomy when combined with either gastrojejunostomy or excision of the antrum. Dr. Colp, you will recall, admitted an incidence of gastrojejunal ulcer in 15 per cent of his patients upon whom he performed gastrojejunostomy—over what interval of time was that, Dr. Colp?

Dr. Colp:—Within two years.

Dr. Wangensteen:—In January, 1941, Dr. Walter L. Palmer, Lester Dragstedt's colleague at the University of Chicago said in a lecture in Minneapolis at a meeting of the Hennepin County Medical Society that, within five years after gastrojejunostomy performed upon patients having duodenal ulcer at the Albert Billings Hospital, 40 per cent of these patients had developed a stomal ulcer. I have the feeling that the chief role of vagotomy will be to delay this occurrence. The present recurrence rate of gastrojejunostomy in patients in whom vagotomy has been added is about 10 per cent—a circumstance which suggests that it is not a particularly happy combination of operations.

Has anyone come forward to suggest that insurance companies will accept patients having had either vagotomy and a drainage operation such as gastrojejunostomy or pyloroplasty or excision of the antrum combined with vagotomy— that insurance companies will accept these patients as standard risks? I do not believe they can or that they will. The dissatisfaction of insurance companies with discarded operations such as gastrojejunostomy is that they failed to protect against recurrence. On the contrary, in my area, because of the demonstration that an effective gastric resection does afford lasting protection against recurrence and thwarts the serious risk of hemorrhage—because of this insurance companies in my area have given insurance at standard rates to such patients 5 years after the operation. I feel that this is an important consideration. Moreover, many patients with peptic ulcer seem to think so too.

Four decades have passed since Edkins (J. Physiol., **34**:133. 1906) described gastrin. The small gastric resection for peptic ulcer had its origin in that suggestion—that elimination of the humoral influence of gastrin would prove an effective operation in combatting the ulcer diathesis, which of course it has not. Surgeons were quicker in recognizing the ill consequences of antral exclusion in their patients than in experimental observations upon dogs, although Koennecke (Arch. f. klin. chir., **120**:537, 1922) had shown that implantation of an antral segment on the ileum invited stomal ulcer. Eiselsberg (Arch. f. klin. chir., **50**:919,1895)

described antral exclusion primarily as a manner of dealing with "inoperable" gastric cancer; however, in the initial report, two cases of duodenal ulcer were included. Haberer (Arch. f. klin. chir., 117:50, 1921) marshalled evidence to indicate that antral exclusion, in which the mucosa was left behind in the pyloro-antral cylinder, potentiated ulcer recurrrence. Even so, some surgeons continued to perform antral exclusion as recently as a decade ago. The work of Dragstedt and his associates (Arch. Surg., 63:298, 1951) in indicating that the secretion from isolated gastric pouches is pyramided in antral exclusion has helped to emphasize to surgeons the hazards of antral exclusion—a warning too which the experiments of Smidt (Arch. f. klin. chir., 130:307, 1924) first gave evidence of a quarter of a century ago.

Sauvage and Harkins (Surg., Gynec. & Obst., 96:127-149, Feb., 1953) in attaching the dog's antrum to the esophagus, suggested that vagotomy lessened the potentiating effect of antral exclusion in the production of stomal ulcer. Some experiments performed in this clinic with an additional control appear to indicate quite definitely that attachment of the antrum to the esophagus is *solely* responsible for the high incidence of occurrence of stomal ulcer which follows this procedure. In an experimental study in which many types of gastric resection were performed, Kelly et al (Proc. Surgical Forum, 4:339, 1953) from this Clinic noted that even extensive gastric resection (80 per cent) was followed by stomal ulcer quite commonly if antral exclusion was performed.

In recent years, there has been a preferment expressed by many surgeons for the Billroth I operation. One of the limitations of this operation, of course, is in the circumstance that many duodenal ulcers with craters do not lend themselves to the performance of the Billroth I operation for technical reasons. In such instances, I would perform tubular resection. For antral ulcer, I feel the Billroth I operation has its most useful indication in the management of peptic ulcer. To have the gastric contents from the residual gastric pouch poured out over the duodenal mucosa is physiologic and is very much to be desired. A number of years ago, Mann and Kawamura (Ann. Surg. 75:208, 1922) observed the occasional occurrence of stomal ulcer following excision of the duodenum in dogs. My associates Drs. Edwin L. Brackney and Alan P. Thal (Proc. Soc. Exper. Biol. & Med., 88:302, 1955) confirmed this observation and noted further that excision of the duodenum in the dog, together with a short segment of the proximal jejunum, was followed by a considerable increase in volume as well as acidity of secretion from an isolated gastric pouch.

These presentations which we have heard today by experimentalists and clinicians have been very informative. Such interchanges should happen more frequently in discussions of the peptic ulcer problem. No one has yet found the ultimate remedy for peptic ulcer. Mark Twain once said he had known many persons who professed to be earnest seekers after the truth, but that it was his experience that most persons when they found what they wanted, abandoned

the search. One reason I have been continuing my search for a better operation for peptic ulcer is in the hope of finding one which will prevent ulcer recurrence and be wholly acceptable from the patient's point of view.

Dr. Seymour J. Gray:—First of all, I should like to congratulate the committee who organized this excellent program. I think it is really an amazingly superior one, particularly in respect to the chairmen and the two coordinators whom we have today—I don't know where you could get two people with such rich experience, who can discuss any subject with broad vision and insight. It is worth the price of admission alone to sit and listen to these two sound off. It has been a wonderful experience.

Of course, I agree entirely with Dr. Snapper regarding the discussion this morning relating to the adrenal steroids. The title of the presentation was not "A New Cause of Peptic Ulcer"; it was "Hormonal Influences Upon the Stomach". The mystery still remains. I think we are chipping away at it little by little.

The adrenal steroids probably do play a significant role in the pathogenesis, but certainly they are not the entire answer. There are a number of other factors involved such as vascular influences, local tissue resistance, and the role of mucus as a protector. For example, we are finding that ACTH causes a fall in the mucus of the gastric juice and this mucus may be related to the protection of the lining of the stomach. We know that peptic cells degenerate when the adrenal is removed from animals, so that the adrenal steroids have something to do with peptic activity.

Then there is the mystery of localized peptic ulceration. Why doesn't the whole stomach slough? Why get one localized area? Vascular disturbances may be one of the answers.

I don't think a comparison of the effects of ACTH upon peptic ulcer and ulcerative colitis is quite fair in that ulcerative colitis represents an inflamatory reaction, and ACTH, as we know, diminishes tissue resistance to inflammation, the fibroplastic response decreases, the inflammatory response to injury is inhibited and there is a decrease in the number of white cells and the leucocyte response, as well as a decrease in gamma globulin and tissue resistance to infection.

Ulcerative colitis is primarily an inflammatory reaction while peptic ulcer is not, but I agree with Dr. Snapper that the ulcer problem involves additional factors too. We still have the old adage however: "no acid, no ulcer", although I think we have failed to study the role of pepsin adequately. Most of the surgical studies in experimental animals, and most of the human studies do not take into account pepsin, which is an important enzyme and does a lot of damage; all the acid may do is present the proper medium for peptic digestion of the tissue.

I should like to say a word briefly about the Mann-Williamson dog, to emphasize Dr. Snapper's remarks. For all practical purposes the Mann-Williamson

dog is a snare and delusion in translating physiological and pharmacological studies to clinical application in humans. Enterogastrone cured the Mann-Williamson dog, but was an utter failure in human peptic ulcer. Kutrol was effective in a Mann-Williamson dog and is apparently ineffective in humans. ACTH and cortisone prolong life in Mann-Williamson dogs, primarily because the dogs live longer as a result of increased appetite and improvement of their nutrition, but ACTH and cortisone produce ulcers, hemorrhage and perforation in humans as well as in experimental animals. One must be exceedingly careful in transferring animal data to clinical use.

I would predict that the patients who have had vagotomy and subtotal resection for the treatment of ulcer will develop the same incidence of marginal ulcers as the nonvagotomized patient, if Dr. Colp does his study ten years from now. Although vagotomy may delay the appearance of the ulcer, it should not prevent its eventual occurrence if the adrenal steroid mechanism takes places. For example, the incidence of marginal ulcer following gastroenterostomy is the same, with or without vagotomy, as Dr. Colp has shown. In the subtotal resection group with vagotomy, the incidence of ulcer is less for this period of study, but, this is a brief study, since 1946. If the study is made ten years hence, the incidence of ulcer may be the same with or without vagotomy. We are seeing an increased number of patients who have recurrent ulcer after subtotal resection and vagotomy.

One must be certain that the level of subtotal resection is the same in both groups. Dr. Wangensteen has been responsible for emphasizing the importance of high gastric resection in preventing recurrent ulcer.

I should like to point out that the massive hemorrhage and perforation that Dr. Wangensteen so beautifully discussed is part of the picture of the ulcers that develop during cortisone therapy. In humans and animals massive hemorrhage and perforation may occur following ACTH or cortisone administration.

We still have to find out why some people respond differently to stress than others. It may be the adrenals or it may be that the stomach is different. The Indonesians and South African Bantus have a low incidence of ulcer. They did live in a very primitive "civilization". Theoretically, what we call "civilization" has taken over in South Africa, and the South African Bantus are getting ulcers. In other words, they are now being exposed to "civilization" and perhaps if the ulcer incidence is studied a few years hence in Indonesia after "civilization" has moved in on them, the incidence of ulcer may be high there, also.

Dr. Ralph Colp:—I should like to thank both Dr. Snapper and Dr. Wangensteen for their discussion, also Dr. Gray.

I wish to state, in answer to some of your questions, that both these series were carried on concomitantly during the years 1946 through 1952. I certainly am mindful of the fact that recurrences can take place. I made that point quite clear. I wish I could be here ten years from now to tell you what our results

will be, but it seems at present that the operation from which we get the best results is this combination of subtotal gastrectomy, which, by the way, is carried on the same way it was done prior to the time of vagotomy—(we are not doing hemigastrectomy), removing from three-quarters to two-thirds of the stomach and accompanying it with vagotomy.

Dr. Frank P. Brooks:—We seem to have agreed on one thing, at least, and that is that you cannot transfer the results of experimental animals to humans and, of course, I don't think anyone would seriously suggest that you pattern your treatment of peptic ulcer on the basis of the results that were shown this afternoon. I submit, however, that it is not always easy to evaluate the effects of therapeutically effective medical therapy in the treatment of patients. I find that it is of considerable assistance to me, and also it is far easier, to design a controlled experiment with experimental ulcers, than it is to rely on some of the reported treatment programs in human peptic ulcers, and it is rather reassuring to be using a drug or program that has been shown to have a definite pharmacologic or physiologic effect, whereas, what one may be giving a patient may actually be in the essence of a placebo.

SURGERY OF THE STOMACH AND DUODENUM IN THE AGED*†

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INTRODUCTION

The population is aging. At the turn of the present century 4.1 per cent of the American people were over 65 years of age. By 1920, though the total population increased by almost 30,000,000, only 4.3 per cent were over 65. In the ensuring 30 years, however, the proportion of inhabitants over the age of 65 almost doubled to the 1950 figure of 8.2 per cent. This ratio continues to increase.

As the population ages the contemporary surgeon is required, with increasing frequency, to deal with disease in elderly people. The experience gained in work-

TABLE I

AGE

Age Group	No. of Patients
60-65	29
65-70	14
70-75	11
75-80	7
80	1
TOTAL	62

ing with the aged should alert him to the fact that the decisions he makes are more gravely significant than the corresponding decisions in younger patients.

Recently a 79-year old man underwent a successful total gastrectomy for the third recurrence of carcinoma of the stomach over a 20-year period. His first resection was done in 1934 when he was 60 years old. This case, among others, has aroused interest in this study of mortality and longevity in aged patients requiring major surgery.

PLAN OF STUDY

In order to clearly present the authors' experience in this field, a study has been made of the 62 patients over the age of 60 (Table I) who have experienced major surgery of the stomach and duodenum during the 10-year period from 1941 to 1951. Gastrostomy has not been considered. In order to demonstrate the

TABLE II

INDICATIONS FOR SURGERY

Indication	Aged	Control
Suspected Carinoma	41%	15%
Pyloric Obstruction	20%	17%
Bleeding	27%	26%
Perforation	9%	30%
Intractability	3%	12%

results in bold relief there have been chosen as controls an unselected group of 103 patients between the ages of 30 and 50 who have undergone comparable surgery within the same time period. The mean age in the group of patients studied was 67 and that in the control group was 40—a difference of 27 years in mean age.

TABLE III

PREOPERATIVE EVALUATION OF THE RISK
(Aged Group)

Risk	Percentage	Mortality
Good	11	28%
Fair	42	15%
Poor	47	41%

DISCUSSION OF DATA

The plan of study has been gauged to eliminate all comparative factors except age from the two groups to be studied so that any difference in the results can be reasonably attributed to the 27-year gap in the mean age difference. All patients in these two groups have been handled under comparable circumstances both with regard to pre- and postoperative care as well as at surgery. The material is from the indigent of Baltimore City. Surgery of the stomach and duodenum has been chosen as reference material because in so doing the element of judgment in deciding whether or not to operate has been reduced to a minimum. A study

of Table II will show that indications for surgery were absolute, no special judg-
ment being required. It is presumed that all patients with these indications were
operated upon regardless of age, so that little or none of the poor results could
escape from inclusion in these statistics on the basis of a decision not to operate.
This would not be true had more purely elective surgery been considered. The
excellent studies of Mithoefer and Mithoefer[2] on the same problem, in which
they used a control, took into account all types of surgery, thereby eliminating
from consideration those patients which were not operated upon because they
were bad risks. Their results, however, were similar to those indicated here in
that they found the mortality rate in patients over 70 somewhat in excess of
four times the mortality rate in patients below that age.

TABLE IV

PREJUDICIAL FACTORS DETERMINING THE RISK (TABLE III)

Factor	No. of Patients
Shock	21
Cachexia	16
Severe Cardiovascular Disease	22
Severe Emphysema	10
Psychosis	5
Hemiplegia	4
Electrolyte Disturbances	7
Severe Alcoholism	3
Obesity	3
Coexistent Carcinoma Elsewhere	3
Far Advanced Tuberculosis	3

A study of Table V indicates that the magnitude of surgery in the two groups
studied is comparable, thus eliminating from the comparison another deceiving
factor. The one patient included who actually had no surgery done died during
the induction of anesthesia. At autopsy there was found a gastric ulcer eroding
into the left gastric artery. This case is included because it seems to properly
belong among the operative mortality patients.

An attempt was made to classify the patients preoperatively as good, fair
or poor risks and then to compare the mortality in these groups (Table III).
Many factors (Table IV), but not age, were taken into account in deciding that
89 per cent of the patients over 60 were less than good risks. The striking informa-
tion herein gained is that when the mortality rates are considered in relation to
the preoperative estimate these are, statistically, little better than a blind guess!

It frequently happens that the good risk dies and the poor risk survives. It is therefore unwise to refuse surgery to the aged patient who is a bad risk at a time when surgery is clearly indicated. Within the past two years five feet of gangrenous small bowel were resected in a moribund patient 68 years of age. This patient was so near to death that no anesthesia was needed for the operative procedure. He died four months later of entirely unrelated causes.

MORTALITY

The results in terms of mortality can be visualized in Table VI. Of the 17 patients who died, autopsies were obtained in seven instances. In the remainder, the causes of death were known or presumed.

TABLE V

SURGERY

Surgical Procedure	Aged	Control
Total Gastrectomy	8	2
Subtotal Gastrectomy	40	68
Closure Perforation	6	31
Gastroenterostomy with Vagotomy	2	0
Gastroenterostomy (Carcinoma)	4	0
Transthoracic Vagotomy	0	3
Exploration Only (Carcinoma)	2	1
No Surgery (Death in O.R.)	1	0
TOTAL	63	105

The overall mortality rate was more than five times as high in the aged as in the control group of patients. In both groups the operative mortality in operations for malignant lesions greatly exceeds the operative mortality for benign lesions. In the present study this is largely accounted for by the fact that many patients were explored when they had far advanced lesions and, even though no major procedure was carried out, they continued to deteriorate and die of their disease within the ensuing month or six weeks. In fact, the operative mortality for exploration alone in patients who had carcinoma has been 100 per cent. This figure is in keeping with the policy of palliative resection whenever possible and gastroenterostomy when obstruction is either present or impending.

The absence of mortality in the perforation of ulcers in the aged is statistically not significant. It is evident that some mortality must certainly accrue in a larger series. Gilmour[5], for instance, in 243 cases of perforation in patients of all ages found an overall mortality rate of 6.3 per cent.

Operative mortality in bleeding in the aged is also five times greater than that in the control group. This is in general accord with the recently published work of Atik and Simeone[1] in which they report a mortality of more than 50 per cent in patients over 50 who have massive upper gastrointestinal bleeding as compared with less than 25 per cent mortality in patients under 50 years of age. It must be agreed that earlier intervention is indicated in elderly people with upper gastrointestinal hemorrhage and that the mortality, ideally, on medical management should be zero. If the patient dies under such management it is certain surgery has been too long delayed.

With regard to total gastrectomy, the mortality rates in this series indicate that total gastrectomy carries in the aged a much higher mortality rate than the subtotal procedure. This wound tend to support the opinion of Larstroell[a] who, on finding mortality for total gastrectomy to be 51 per cent in 54 cases, decided that total gastrectomy is not worthwhile in patients of advanced age because the results are too discouraging.

TABLE VI

MORTALITY

Mortality	Aged	Control
Overall Mortality	27%	4.8%
Benign Lesions	20%	4.7%
Malignant Lesions	46%	20.0%
Perforation	0	0
Bleeding	35%	7.0%
Subtotal Gastrectomy	20%	5.9%
Total Gastrectomy	37%	0 (2 cases)

In considering the mortality results in each category enumerated in Table VI, it is indicated clearly that mortality subsequent to major surgery is four to five times as high in the aged group of patients as compared with the controls. With the elimination of other factors it is obvious that this difference must be attributed to old age with its attendant degenerative processes.

SURVIVAL

Of the 62 patients studied, 17 are listed as operative mortalities and eight others could not be followed. Of the 37 remaining patients followed for from three to ten years, the average survival time to date has been three and a half years. Many of the patients are still alive and well. It should be mentioned for comparative purposes that at the age of 67, the mean age of these patients, the

statistical life expectancy is not less than ten years. No comparison has been drawn between the average survival time of the aged and control groups of patients since these figures are, for obvious reasons, not comparable.

The average survival time for patients operated upon for carcinoma has been two years, with no five-year survivals to date.

TABLE VII

CAUSES OF DEATH

Principal Cause	No. of Patients
Terminal Carcinoma (operated upon)	5
Bronchopneumonia (autopsied)	3
Bleeding Esophageal Varices	1
Exsanguination during Induction of Anesthesia	1
Pulmonary Edema (2 autopsied)	3
Blown Duodenal Stump with Peritonitis	2
Sudden, Unexplained, 10th Postop. Day Following Subtotal Resection	1
Sudden, Unexplained, 11th Postop. Day Following Total Resection	1

ASSESSMENT OF RESULTS

Patients operated upon for carcinoma tended to live tolerable lives during the first half of their remaining time but to be progressively more miserable during the latter half. This type of result has been considered only fair although the duration of life may have been prolonged considerably by the operative pro-

TABLE VIII

RESULTS OF SURGERY

Result	Percentage
Good	46
Fair	21
Poor	33

cedure. Patients with operative complications causing prolonged convalescence or permanent disability are noted as poor results. Operative mortality is included under poor results. An assessment of the results, in terms of surgery, is shown in Table VIII.

The final analysis, however, is still incomplete. In assessing the results it seems clear that the duration of life following surgery is of much less real significance in the aged than is the nature of the life led. Old people desire comfort more than they want or need prolongation of their lives. It is the physician's obligation to the aged, as to the young, to prolong life not only as a vegetative object but also in terms of comfort and happiness. Some of the 46 per cent of patients included in the good result group have spent their remaining years in a state of senility or plagued by degenerative illnesses distinctly apart from the surgical lesion. It seems only fair in making a complete summary to take these unhappy circumstances into account. Judged with all factors in mind, both surgical and nonsurgical, it was found that 35 per cent of the 54 patients followed had good results from a surgical point of view and subsequently spent their remaining years in comfort—completely able to enjoy life.

Conclusion

There is no substitute for good judgment. Statistics of the type herein presented are of no real value in considering the individual patient for completely elective surgery. There is often a vast difference, for instance, between the numerical age and the physiologic age of a patient.

Good judgment, however, must be based, in individual cases, upon a knowledge of the facts. The facts, as indicated by the present study are:

1.   The mortality rates for major surgery in the aged are significantly higher than in younger people.

2.   It is impossible at present to predict which patient can survive a certain procedure and which one cannot. Therefore, when the lesion is a mortal threat one has no alternative but to carry out the surgery necessary to preserve life.

3.   In the aged, the accomplishment of a good surgical result is not necessarily a service to the patient. The senile and the psychotic, for instance, should probably not be given extensive surgical treatment unless the lesion in question is threatening life.

4.   It seems fair to speculate that a consideration of a more purely elective type of surgery of the stomach or duodenum of the aged patient would reveal a higher percentage of better results, both surgical and sociological.

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Discussion

Dr. I Snapper:—Dr. Brantigan's paper is a very timely one because not only physicians but also surgeons are nowadays so frequently called upon to solve geriatric problems. I fully agree with Dr. Brantigan that old people do not tolerate operations as well as young people. If a geriatric *service* patient has to be operated on, I always add to the transfer note "This patient should be operated on by the chief." Needless to say that resident training is absolutely necessary and that occasionally a resident must be entrusted with an operation for which he is not yet completely prepared. In such cases the risk for the patient is reduced as much as possible when an attending surgeon assists at the table.

Dr. O. H. Wangensteen:—The mean age of our patients with gastric cancer has been about 65 years. As Dr. Brantigan said, age does make a difference. My colleague, Dr. Clarence Dennis, now of Brooklyn, reported eight successful successive resections for nonviable intussusception in infants. However, when resection was necessary, for strangulated hernia, Dennis and Varco of our clinic had a mortality of more than 10 per cent—because of the factor of age alone.

Oliver Wendell Holmes said that at age 50, old age calls on us and leaves his calling card. Most of us reject it, but at age 60, I suspect most of us will be inclined to accept the old man's call. However, a few protest the old man's visits up to the biblical three score and ten.

In a sense, Dr. Brantigan's experience only proves the validity of the old biblical admonition "that the years of man are three score and ten, and if by strength they be four score years, yet is their strength but labor and sorrow." One of the questions I like to ask every old patient whom I see in the clinic is how old he wants to live to be. Usually their answers are quite the same: as long as the weather is fair and the company is reasonably good, all old men want to keep their frail craft afloat. Seldom do we find it necessary to decline operation in old people because of the consideration of the mortality of age per se. I confess freely that the mortality is larger, but as Dr. Snapper said, these cases need to be managed particularly carefully. One needs to take more care with every item of the management in the surgery of old people. Younger persons have a far greater tolerance to withstand trauma.

One thing pointed up by Dr. Brantigan's presentation is the important necessity of developing good and adequate supervision of our surgical fellows and residents in municipal hospitals. More and more, I believe, it is becoming recognized that the standard of surgery in our municipal hospitals is contingent in a very direct manner on the extent and the expertness of the supervision. In Metropolitan Medical School areas in particular, there exists, I believe, a great opportunity to improve the surgical accomplishment by inviting full-time young surgeons,

ambitious for academic careers in surgery, to supervise the surgical residents; to instruct them in the intricacies of fluid and electrolyte needs of surgical patients. Continuity of effort and of instruction pays elaborate dividends, especially in the care of old patients. The accomplishment in Veterans Hospitals which operate under a Dean's Committee, and the experience of University Surgical Clinics are demonstrating the superiority of supervision of the activities of the surgical service by full-time surgeons with academic leanings. And when the time comes that municipal hospitals in all Metropolitan Medical School areas have full-time surgical supervisors, the quality of the service of the Staff of those hospitals to the patients will be improved. Moreover, the status of those hospitals as training centers for well qualified surgeons will be enhanced. Such a development in our country could do a great deal toward the advancement of surgery.

Dr. Otto C. Brantigan:—It is good to have the last word. I shall have to confess that before we made this study, I had the feeling that chronological age was not really important so far as mortality was concerned. If these old patients were cared for carefully enough, I thought they would do as well after operation as the young ones. The truth of the matter is that our study did not show it.

I am more or less convinced it is a good study since the surgery was done by the same calibre of surgeon throughout the years. The study was restricted to a particular area, the stomach and duodenum, which eliminates the selectivity of patients. Because the mortality apparently is high we were criticized on the grounds that the resident surgeon group is not capable of doing the surgery, but I would take exception to that. These men are at least in their fifth year out of school. Some of them probably do it better than their chief would do it. The study concerns a pretty carefully controlled group of diseases and surgeons. Therefore, I think that we must face up to the fact that the older individual does not do as well as the younger adult and his diseases of the stomach are different from those of the young adult. I wish to repeat that in the aged group 36 per cent of the operations were emergency procedures done for perforation or bleeding and therefore had to be carried out in spite of their general condition. Another 41 per cent were operated on for malignancy. This represents consecutive patients with stomach or duodenal lesions without advantage of selection. I want this opportunity to state emphatically that our mortality for elective subtotal gastrectomy for nonmalignant lesions is 1.2 per cent.

You know, there have been reports recently, one by Mithoefer and Mithoefer where they took a very large group of patients including all types of surgery, such as nailing of hips, fractures, hemorrhoids, and so forth, and in such a group there is a very high degree of selectivity between the young and old age group. Nevertheless, their figures showed a mortality four times as great in the older age group as in the younger. They started their old-age group at age 70.

I believe age does influence mortality and I believe it is shown by our well controlled study.

PROGNOSIS AND THERAPY IN CHRONIC HEPATITIS*

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Introduction

During the past 15 years, viral hepatitis has been epidemic in many widely separated areas and has been recognized as a major problem in world health[1]. All age groups have suffered, but attention has been directed predominantly to the effects of this disease on young men of military age. Many of the concepts expressed below were derived from experience with hepatitis in troops and they need not reflect the exact pattern of the disease in other groups. In addition, much of the available data are derived from patients who had hepatitis with jaundice, and not many observations are included on that considerable percentage of patients who failed to develop clinical icterus. These limitations make it necessary to qualify any discussion of chronic hepatitis with the admission that its overall incidence following the acute disease is not known, although its variability in different groups is recognized. For example, attention has been called to the high incidence of the chronic disease in women beyond the menopause[2] in contrast to the low incidence in troops[3,4].

In young men, the course of disease is usually benign, although approximately 15 to 20 per cent continue to have some evidence of hepatic dysfunction for varying periods after the expected time of recovery; most of these patients usually have complete restoration of function within six to eight months. A small percentage of patients (probably less than 1 per cent) develop chronic hepatitis. The exact duration of time before patients with protracted acute hepatitis may be said to have chronic disease is a matter of discussion, since restoration of normal hepatic function and morphologic appearance of the liver has been found to occur as long as two years after onset[5]. When, however, evidence of hepatic disease has been present intermittently or constantly for one year, this diagnosis should be strongly considered.

The course of disease in those patients who fail to make a complete or sustained recovery from acute hepatitis is not easy to predict, and the difficulty is enhanced by the lack of understanding of the conditions which potentiate the development of sequelae. Although it has been frequently suggested that inadequate treatment, preexisting hepatic damage, age over 40 years, severity of acute attack, and the presence of intercurrent infection predispose patients to develop

various sequelae, it is impossible to incriminate consistently any factor which can be defined as having contributed to continued hepatic disease. In addition, it is not known whether variation in strains of virus or persistence of virus in the body may be operative, although the demonstration of hepatitis virus for prolonged periods in the blood of patients with hepatic cirrhosis is of interest in regard to the latter point[6].

Prognosis

The manifestations of residual hepatic disease may range from asymptomatic mild deflection of certain tests of hepatic function to severe debilitating illness progressing to hepatic failure and death. One of the problems in evaluating patients presenting findings suggestive of hepatic disease long after the onset of acute hepatitis is the sharp dissociation which frequently exists between subjective and objective evidence[7,8]. Attention has been called to a wide variety of symptoms occurring in patients who had presumably recovered from hepatitis and whose tests of hepatic function and morphologic appearance of the liver were normal. It is also equally well known that patients may have a considerable degree of histologic alteration of the liver both with and without evidence of hepatic dysfunction or clinical signs or symptoms.

In spite of the inherent difficulties in the prognosis of patients who have had hepatitis and in whom the question of chronic hepatitis is being considered, a correlation of the history with the results of physical examination and various tests of hepatic function frequently serves to define the severity of hepatic disease and to suggest its possible outcome. In certain patients, biopsy of the liver adds valuable information. For purposes of classification and to aid in prognosis, these patients may be divided into the following three groups: (I) patients with no disease, (II) patients with residual dysfunction but without apparent progression of disease, (III) patients with chronic hepatitis.

Group I. Patients with no disease:—A certain percentage of patients continue to complain of easy fatigability, anorexia, intolerance to fat and alcohol, discomfort in the right upper quadrant, and emotional instability long after ostensible recovery from hepatitis. The liver may be palpable but is usually not tender. A recent study by Nelson et al[9] among U. S. Occupation troops in Germany revealed that the occurrence of symptoms, signs, or slight abnormalities of hepatic function was not uncommon in a group of 80 men who were doing full duty and who were re-examined two to three years after recovery from hepatitis. The lack of correlation between the occurrence of complaints and objective findings, however, as well as their mild character, made it impossible to assign them any significance. Under such circumstances and in the absence of any significant hepatic dysfunction or histologic changes in the liver, there is no reason to attach any unfavorable import to these findings nor to consider them as evidence of chronic hepatitis.

Group II. Patients with residual dysfunction but without apparent progression of disease:—The most frequently occurring abnormalities of hepatic function following hepatitis are persistent or recurrent hyperbilirubinemia and abnormal retention of intravenously injected bromsulfalein. Patients in this group are usually asymptomatic, although on occasion they may complain of mild discomfort in the right upper quadrant or excessive fatigue after indulgence in violent exercise or alcohol. The liver may be palpable but is usually not tender. Histologic examination of the liver frequently reveals scattered tiny areas of necrosis with infiltration by round cells, fatty vacuolization of the hepatic cells, and slight increase in periportal connective tissue. These changes are usually so mild that it is difficult or impossible to distinguish them from the histologic alterations occasionally found in patients without hepatic functional abnormalities. Although the amount of information on long-term observation is not great, the available evidence suggests that the prognosis in these patients is good and that the functional or histologic changes or both together, if mild, apparently may persist for years and be entirely compatible with full activity and without apparent deleterious effect. Serial observations are required at frequent intervals to define the status of such patients.

Group III. Patients with chronic hepatitis:—The association of many or all of the above-mentioned symptoms and varying degrees of abnormality of hepatic function, with or without enlargement and tenderness of the liver, is found in these patients. Splenomegaly may be present. The histologic alterations of the liver are variable and have been described as postnecrotic scarring, multiple nodular hyperplasia, primary biliary cirrhosis and, uncommonly, portal cirrhosis. The degree of hepatic disturbance is variable both as to intensity and rate of progression. It apparently may be arrested and remain inactive with fixed residual dysfunction for varying periods of time, only to become active again subsequently. It is believed that, on occasion, prolonged disease may eventually culminate in recovery; however, the usual pattern is one of progressive hepatic insufficiency.

It is often difficult to decide whether the evidence obtained from the history and physical and laboratory examinations represents residual hepatic dysfunction at a fixed level which will improve, or a phase of progressive disease which will inevitably become worse. In addition, it is impossible to predict the rate of progression of active chronic hepatitis or whether ostensibly inactive disease will be reactivated at some time in the future. The clinical and histologic studies of sufficient numbers of patients over a period of years necessary to answer these questions are not yet available.

At present, it is believed that patients in this group who are without symptoms and hepatic tenderness and whose hepatic dysfunction is apparently fixed over a period of several observations may be regarded as having little or no activity of disease. In contrast, progression of disease is commonly manifested by persistent or recurrent jaundice, strongly positive thymol turbidity tests, the

appearance of *spider naevi*, loss of libido, and failure to gain in weight. Abnormal retention of fluid resulting in edema and ascites, hemorrhage from ruptured esophageal varices associated with portal hypertension, and hypersplenism are frequent complications and of serious nature.

THERAPY

Group I:—No therapy is required for these patients except the reassurance that they do not have hepatic disease.

Group II:—Therapy consists of providing a regimen of good hygiene and a well-balanced diet, reassurance, and the avoidance of excessive fatigue. Careful attention to intercurrent infections is recommended.

Group III:—The care of these patients is difficult and not infrequently disappointing. In contrast to its effect in Laennec's cirrhosis, the provision of an excellent, well-balanced diet is of less striking benefit in chronic hepatitis, although it is recommended as it may improve to some degree the general nutrition. The use of supplemental lipotropic substances such as methionine or choline has not been found to be of value. Rest is important, but its duration and degree are poorly defined. Too often, patients are kept in bed for prolonged periods apparently without sufficient reward in improvement to warrant such therapy. As in the case of any chronic disease, the values and disadvantages of a state of invalidism must be considered in each patient. More often than not, it is far better for the patient to be engaged in some occupation, whole or part-time depending on the phase of disease, which will contribute to his livelihood and interest in life. Those patients who are not believed to have activity of disease may lead essentially normal lives, with careful attention to rest, diet, and early therapy of intercurrent infections.

Certain patients, if treated early in the course of chronic hepatitis, appear to be benefited at least temporarily by the administration of one of the broad spectrum antibiotics such as aureomycin or terramycin in doses of 250-500 mg. every six hours for two to four weeks. These materials at times, however, seem to be of more value in the care of patients with exacerbations of activity of disease. The use of ACTH or cortisone may also be of some value in initiating progressive recovery in some patients if treated early in the course of chronic hepatitis, particularly in the cholangiolitic type, although it is generally agreed that such therapy, once started, must be continued until maximum improvement has occurred.

Retention of fluid:— It has been suggested that, in contrast to the situation in patients with Laennec's cirrhosis in whom retention of sodium is often among the most important factors in potentiating the accumulation of ascites, hypoalbuminemia may be more important in patients with chronic hepatitis. If the serum albumin is diminished, the intravenous administration of salt-poor human albumin, in amounts of 25 grams per day, at variable intervals, depending on

the patient, may be of assistance in limiting the retention of fluid for prolonged periods. Restriction of sodium in the diet is of assistance in those patients who have impaired capacity to excrete it.

Portal hypertension and hypersplenism:—Hypersplenism may be relieved by splenectomy; however, in the presence of portal hypertension and hemorrhage from ruptured esophageal varices, splenectomy with splenorenal shunt is the procedure of choice. If hypersplenism is not present in a patient with bleeding varices, portacaval anastomosis is preferred since it is likely to give a more effective reduction of portal pressure. The criteria of fitness for operation are difficult to evaluate. The presence of ascites is a recognized contraindication and reveals the necessity of improving the general physical condition before operation. In addition, serious abnormality of various tests of hepatic function constitutes a contraindication. It is difficult, however, to predict on the basis of tests of hepatic function what a patient can tolerate, and in each case it is necessary to have as a basis for decision a thorough appreciation of the general clinical status as well as the results of functional studies of the liver. Since hemorrhage from a ruptured varix may be fatal, may precipitate hepatic failure, or may impair hepatic function to such a degree that operative intervention must be deferred for a considerable period, numerous studies are now being conducted on the advantages of prophylactic shunting procedures before hemorrhage occurs in such patients.

SUMMARY

A variable percentage of persons who contract acute hepatitis have symptoms, signs, or hepatic functional abnormalities for considerable periods after the expected time of recovery. Most of these have (a) symptoms unassociated with other objective changes (Group I) or (b) mild hepatic dysfunction which is apparently not progressive (Group II). A very small percentage of patients have chronic hepatitis (Group III) which may progress to hepatic insufficiency, with the development of any one or all of the following complications: (a) portal hypertension, (b) ascites, (c) hypersplenism, (d) hemorrhage from ruptured esophageal varices. Therapy for the first two groups of patients is largely concerned with good hygiene and reassurance. Therapy for those who develop chronic hepatitis is directed to the provision of adequate diet, rest, and the care of the various complications which arise.

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<center>DISCUSSION</center>

Dr. Emil Gribovsky (Kingston, Pa.):—I should like to ask Dr. Havens a question. How many of the chronic hepatitis cases develop cirrhosis, and also whether alcoholic intake has something to do with the chronicity?

Dr. I. Snapper:—In every patient with hepatitis the danger of the development of cirrhosis must be considered. In weighing this possibility I follow certain rules. If a patient develops ascites during hepatitis, he is in my experience a candidate for a postnecrotic cirrhosis. I know that during World War II many military physicians in the field saw patients with hepatitis and ascites who recovered completely. My personal experience in this country and also in the Old World is different and nearly all my patients who developed ascites during hepatitis returned later with postnecrotic cirrhosis. Therefore I consider the development of ascites during hepatitis as a prognostically unfavorable sign. I also feel that the outlook for complete recovery is less favorable when in the course of hepatitis splenomegaly develops.

The splenomegaly has no prognostic significance in the case of a hepatitis developing during mononucleosis.

Increase of the serum globulin occurring during hepatitis is a serious sign. All patients with hepatitis and a marked increase of globulin are in great danger of a postnecrotic cirrhosis.

There is no reason why a high protein diet which has a favorable influence in Laennec's cirrhosis should improve the outlook of hepatitis. Laennec's cirrhosis occurs in areas where the protein intake is low, i.e. in many Oriental countries. In many of these areas hepatitis is endemic and the course of this disease is usually favorable. In other words, the same Orientals who easily contract Laennec's cirrhosis, suffer from hepatitis without developing yellow atrophy or postnecrotic cirrhosis. It should be added that Laennec's disease is closely connected with alcohol intake and/or with low protein intake, whereas postnecrotic cirrhosis

has a completely different etiology. All these points indicate that there is no reason at all why the treatment of hepatitis and Laennec's liver cirrhosis should be the same.

I know only one drug which is useful in the initial stages of chronic hepatitis. If in a hepatitis the jaundice does not clear up, the liver functions deteriorate, the serum globulin increases and the patient complains about excessive fatigue and sleepiness then, as Dr. Havens has mentioned, intravenous injections of salt-free albumin often seem to improve the condition and may stave off a hepatic coma.

One has the impression (a) that the injected serum albumin collected from healthy persons, is superior to the albumin manufactured by the diseased liver cells; (b) that the normal serum albumin is a favorable factor for the regeneration of the diseased liver cells.

In patients with esophageal varicosites which develop in the later stages of postnecrotic liver cirrhosis, intravenous serum albumin should not be given. Such injections increase the circulating blood volume and thereby increase the danger of a hemorrhage. Many cases of Laennec's cirrhosis which never had had a hemorrhage, started to bleed from varicosities after intravenous albumin injections. These albumin injections are therefore indicated in chronic hepatitis when the jaundice persists, the liver function deteriorates, pruritus becomes intolerable, but no varicosities have developed yet.

Dr. W. Paul Havens, Jr.:—In response to the question of how often does chronic hepatitis develop into cirrhosis, I think it only fair to say that confusion in terminology makes it difficult to give an acceptable answer. It is not easy to find unanimity of opinion in any group of pathologists concerned with defining the histologic alterations found in the liver of patients suspected clinically of dying with postnecrotic scarring. Although a considerable diversity of histologic alterations may occur in chronic hepatitis, the predominant changes characteristic of postnecrotic scarring are those which follow necrosis of large areas of the hepatic parenchyma with collapse of the reticulum, eventual regeneration of cells, and resulting distortion of the lobular architecture. The nature of these changes makes it possible to find areas of normal lobular architecture in contrast to the situation in patients with so-called Laennec's cirrhosis in whom, in well-advanced disease, it is unusual to find any normal lobular relationship.

The role of alcohol in the transition from acute to chronic hepatitis is unknown. There is little evidence that it is of any importance and, in reviewing the follow-up records of large numbers of troops who were not reluctant to drink alcoholic beverages, after hepatitis or even during its course, it is difficult to find convincing evidence of damage that could be attributed to their indulgence. In spite of this and in view of the lack of knowledge on the possible bad effects of alcohol on an already damaged liver, it would seem appropriate, however, to advise abstinence for a short period following recovery.

I share Dr. Snapper's concern for the patient who, during the acute phase of hepatitis, develops a very high serum globulin which persists into the period ordinarily regarded as convalescence. There is a striking difference between the considerable degree of hypergammaglobulinemia which develops in patients with mononucleosis and with hepatitis and the mild elevation in gamma globulin in patients with many other infectious diseases. This suggests the possibility that the great increase in globulin in the former diseases may not be wholly a response to an infectious agent but may also be an indication of a response to some other antigenic substance conceivably derived from the products of hepatic damage.

In reply to Dr. Snapper's question concerning the significance of splenomegaly persisting for some time after hepatitis, it seems to me that in such patients one does have reason to suspect the possibility of development of chronic disease. Splenomegaly does, however, apparently, persist as an unusual phenomenon in a certain number of patients who have no evidence of hepatic disease afterward. Dr. Kalk, in Germany, has called attention to this point.

Dr. Snapper has indicated his belief that salt-poor human albumin is a valuable therapeutic aid for patients who are seriously sick in the acute phase of hepatitis. With this I agree. In addition, I think it is of value in patients with chronic hepatitis who have ascites associated with hypoalbuminemia. We have treated a number of such patients with salt-poor albumin and have been able to control excessive retention of fluid for many months and even as long as two and three years. They require varying amounts, ranging from a unit every two to three weeks to a unit two to three times a week. I appreciate and respect the possibility of rupture of varices, and I have had occasional bad experiences, but the value of albumin in such patients often outweighs this risk.

The prognostic value of the occurrence of ascites during the acute phase of hepatitis is not easy to judge, and it occurs in small amounts far more frequently than is recognized. When it occurs in large amounts, it indicates serious hepatic disease; however, I am unable to say that it invariably means that chronic progressive hepatitis will occur. Rather, it would indicate to me that a great deal of liver had been destroyed. The degree of regeneration and the architectural pattern subsequently established are unpredictable.

REGURGITATION ESOPHAGITIS*

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The diagnosis of esophagitis can usually be made on the basis of the following symptoms: dysphagia, high epigastric or substernal pain, heartburn, eructations, and regurgitation. The symptom-complex in severe cases is extremely distressing and persistence of the underlying pathological changes may eventually lead to weight loss, hemorrhage, stricture, hiatus hernia, and even perforation of the viscus. When definite mucosal and other anatomical changes exist the clinical impression is readily confirmed by esophagoscopy and by roentgenological examination.

The acute erosive esophagitis followed by progressive stenosis of the greater portion of the esophagus (Fig. 1), caused by the ingestion of corrosive substances, is so obvious as to etiology and effect that no more time will be given to its discussion here.

An almost identical picture of esophageal stricture may be seen following severe and prolonged vomiting of pregnancy (Fig. 2). The etiology in this situation is quite different but the ultimate effect is similar to that which follows esophagitis due to chemical agents. Increased intraabdominal pressure and the change in position of the stomach and other viscera associated with the enlarging uterus probably influence the onset of vomiting to some extent, but it is believed that regurgitation of gastric contents and vomiting are of primary importance in the development of esophagitis. Intubation for prolonged periods may also contribute to the development of ulcers in an already inflamed esophagus.

There are innumerable reports in world literature concerning the etiology of nonspecific esophagitis and idiopathic stricture in which a wide variety of possible causes is mentioned. In many of these reports regurgitation of gastric contents is mentioned only to be dismissed or relegated to a rather insignificant role as a factor in the production of inflammatory change. Perhaps the reluctance to accept regurgitation of gastric juice into the esophagus as the most important single factor in the production of nonspecific esophagitis is based upon the fact that regurgitation is still considered by many to be a normal physiological occurrence; and also, that esophagitis occurs in conjunction with a wide variety of diseases which in themselves have been considered to be direct causes of esophagitis.

An analysis reveals that nausea and vomiting are symptoms common to many of those diseases. This is true in the acute infectious and toxemic diseases, cholecystitis and cholelithiasis, peptic ulceration of the stomach and duodenum,

head injury, brain tumor, and other diseases of the central nervous system, as well as in the acute diseases of infants, and the generalized debilitating diseases of old people. If we accept as fact the deleterious influence of the action of the acid-peptic factor of gastric secretion on the mucosa of the lower end of the esophagus, then it is not surprising that esophagitis occurs so frequently in asso-
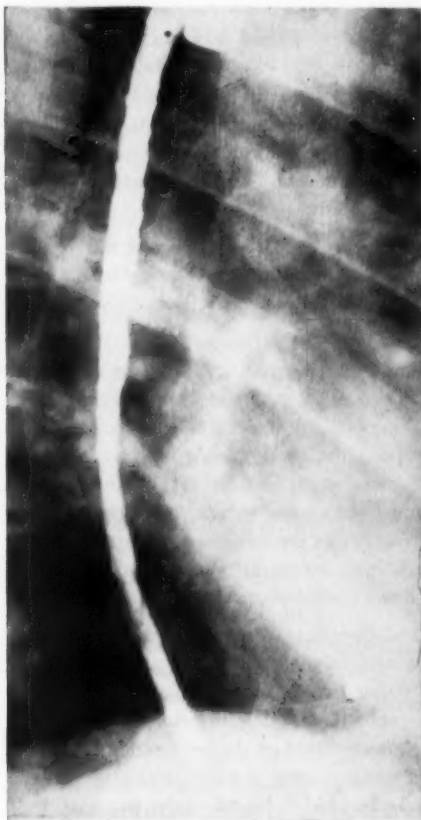

Fig. 1   Fig. 2

Fig. 1—Case 1. Stricture of esophagus in 21-year old male who drank a solution of lye in an attempt at suicide.

Fig. 2—Case 2. Stricture of esophagus in 29-year old white female who developed esophagitis during the course of a severe attack of vomiting of pregnancy.

ciation with diseases in which nausea and vomiting, or regurgitation, are prominent features.

The work of Wangensteen and others[1] at the University of Minnesota seems to have proved rather conclusively, in animals at least, that "contact of acid gastric

juice with esophageal mucosa, whether brought about by vomiting, regurgitation, or by direct application, has a very prompt and devastating effect."

Dawson[2] states that "the term reflux esophagitis denotes an inflammatory reaction by the squamous epithelium in the distal esophagus to regurgitation of gastric juice."

Olsen and Harrington[3] are of the opinion that peptic ulceration of the esophagus may be the result of "(1) excessive or prolonged vomiting, or (2) incompetence of the physiologic sphincter at the cardia, when it occurs in association with hiatal hernia or with reflex spasm of the lower end of the esophagus."

Fig. 3a   Fig. 3b

Fig. 3a—Case 3. Lateral view of esophagus showing stricture and ulcer in lower third.
Fig. 3b—Same case—Lateral view showing large gastrojejunal ulcer.

The association between gastric and duodenal ulceration and esophagitis, with or without stricture, has been noted by Dawson[2], Benedict and Sweet[4], Wangensteen and Leven[5], Deaver and McAlpine[6] and others. Most of them believe that regurgitation of acidic gastric secretion is responsible for the production of inflammatory changes in the esophagus.

The following case (Fig. 3a and b) is an example of a situation in which duodenal ulcer, gastrojejunal ulcer, and ulcer of the esophagus with stricture were found in a 74-year old man on whom a gastrojejunostomy was done in 1926

because of pyloric obstruction. A stomal ulcer was first noted in 1952 and stricture of the esophagus in 1954. Vomiting, regurgitation and heartburn had been rather prominent symptoms during the past year.

Hiatus hernia is perhaps the most frequent diagnosis made on patients referred for x-ray examination because of symptoms of dysphagia, heartburn, and high epigastric or substernal pain. The term hiatus hernia is in many cases loosely applied and may mean either congenital thoracic stomach or herniation of the stomach through the esophageal hiatus. Recent reports indicate that most cases of hiatus hernia are not congenital, but are acquired as a result of repeated vagus stimulation or cicatricial contraction resulting from repeated attacks of esophagitis. This is not difficult to understand when we consider the fact that
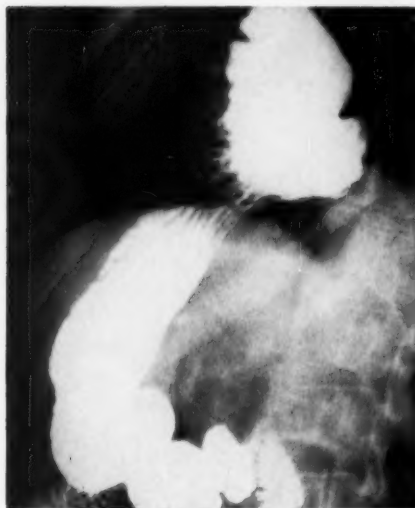
Fig. 4   Fig. 5

Fig. 4—Case 4. Hiatus hernia, probably congenital, in 63-year old white female who com-
       plained of mild dyspepsia for many years.
Fig. 5—Case 5. Relaxed esophagogastric sphincter and hiatus hernia showing mucosal changes
       suggestive of esophagitis in 51-year old white female with symptoms of dull substernal
       pain and gaseous eructations for one year. The symptoms were more severe after
       eating and after going to bed.

hiatus hernia is most common in patients past the age of 50 years and in whom obesity and biliary or gastroduodenal disease may be present.

Dey and others[11], in reporting the results of experiments on dogs, state that "shortening of the esophagus with the production of hiatus hernia due to traction upon the stomach in the dog results from electrical stimulation of the vagus nerve or reflexly from stimuli having their origin in the viscera of the upper abdominal cavity."

Olson and Harrington[3] found in a study of 220 cases of short esophagus that only 4 per cent appeared to be of the congenital type. They found that in the majority of cases an actual esophagitis was present and that cicatrization had produced sufficient traction to draw the cardia of the stomach into the chest.

These and many other reports[7,8] in the literature lead one to the conclusion that regurgitation of acid secretions must play an important part in the produc-

Fig. 6—Case 6. Roentgenologically normal esophagus of 24-year old white male who developed severe symptoms of esophagitis one year previously following meningoencephalitis. He was tube fed for two weeks during acute illness. Metal clip indicates location from which biopsy was taken. Tissue on microscopic examination showed acute esophagitis.

tion of symptoms of hiatus hernia and that it must also play an important part in the production of the associated anatomical changes.

The roentgen diagnosis of hiatus hernia may be made with ease or with difficulty. In many cases of short esophagus (with or without stricture) (Fig. 4 and 5) hiatus hernia may be recognized after the first swallow of barium mixture.

In other cases the hernia can be demonstrated only after considerable maneuvering, with the patient in the supine Trendelenburg position. In these patients forced respiratory movements and the application of pressure to the abdomen may be necessary to show the herniation. In still others, a patulous esophagogastric orifice (Fig. 6) may be the only finding. The nature and degree of the herniation may account for the character of the symptoms, which according to Shanks[9], may be divided into two main groups: those due to the effect of the herniation of the stomach, such as epigastric discomfort, flatulence, belching, regurgitation and vomiting; and those due to the effects of pressure on adjacent thoracic viscera, such as palpitation, anginal pain, dyspnea, and hiccough.
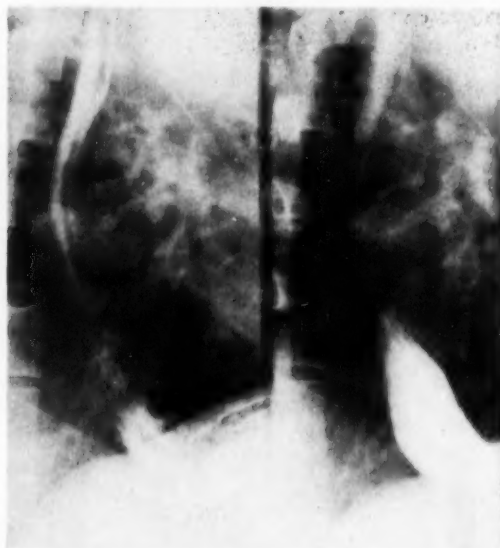
Fig. 7—Case 7. Roentgenologically normal esophagus of 59-year old white male with symptoms of heartburn, distention, and acid eructations for several years. Frequent regurgitation of gastric contents in supine and erect positions was noted during fluoroscopic examination.

Another group of patients may show no definite herniation on fluoroscopic examination but merely rapid and recurring regurgitation of barium mixture into the esophagus. This may occur with the patient in either the erect or the horizontal position.

Regurgitation is not frequently seen during fluoroscopic examination, but when it does occur it is rather sudden and dramatic. Very frequently the fluid returns to the stomach so rapidly that there is not enough time, unless stricture is present, to record the phenomenon on a spot film of the region. It is not likely that regurgitation during fluoroscopic examination will reproduce symptoms

unless acute ulcerating esophagitis or ulceration with stricture is present. This type of regurgitation occurs without warning and should not be confused with regurgitation which is occasionally seen in patients who are made nervous or apprehensive by the examination or who become nauseated by the taste and feel of the barium mixture. This is, in my opinion, very significant and may be sufficient reason, in many cases, to warrant a presumptive diagnosis of esophagitis. It may also be sufficient evidence upon which to base a prediction that esophageal stricture, hiatus hernia, or both will eventually occur. Regurgitation can be observed only during fluoroscopic examination and every effort should be made on the part of the radiologist to elicit this phenomenon in patients suspected of having esophagitis or hiatus hernia. It is the responsibility of the radiologist to report the phenomenon when it is observed during fluoroscopic examination in order that it can be properly evaluated by the referring physician.

Recent literature on the subject of esophagitis places more and more emphasis on regurgitation as the prime factor in the production of esophagitis. Wangensteen[10] in "An Assessment of the Etiologic Aspects of Peptic Ulcer and Surgical Therapy" concludes that "1. Idiopathic stricture of the esophagus is acid-peptic ulcer or disease of the esophagus" and "2. Spontaneous perforation of the esophagus in man is in all likelihood acid-peptic perforation of the esophagus." If these views are generally accepted, is it possible that there will be a change in the surgical treatment of esophagitis, stricture, and hiatus hernia? Will vagotomy and gastrojejunostomy, or gastric resection be the operations of choice, reserving the direct attack on the diaphragm only for those cases in which the pressure group of symptoms is predominant? Will the roentgen observation of regurgitation of gastric contents into the esophagus lead to more active and insistent therapeutic measures on the part of the gastroenterologist?

It would seem that medical and surgical treatment should be directed more toward the control of factors which influence the production of esophagitis, stricture, and hernia, rather than primarily toward the anatomical deformities which have resulted from inflammatory disease of the esophagus.

To this date very few cases requiring surgical therapy have been treated by the more radical departures from accepted methods of repeated dilatation for stricture and diaphragmatic repair for hiatus hernia. Perhaps more knowledge of the subject will be forthcoming during the next few years.

SUMMARY

In this discussion some of the recent literature on the subject of regurgitation esophagitis and its sequelae has been reviewed. The importance of the roentgenoscopic observation of regurgitation of gastric contents into the esophagus has been mentioned as constituting sufficient evidence upon which to base a presumptive diagnosis of esophagitis.

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Discussion

Dr. I. Snapper:—The problem of esophagitis is a very important one, but the problem of acid digestion and ulcer formation may be somewhat different.

After the first trials to visualize stomach and esophagus with barium meals had been successful, the first atlas of roentgenology contained photos of the frequent combination of pylorospasm and cardiospasm. It has gradually become evident that, if in a patient these two anomalies are found or, if a patient has an ulcer of the duodenum, and at the same time cardiospasm, esophagoscopy will show the presence of a peptic ulcer of the esophagus.

Anyone who suffers from an ulcer of the stomach or duodenum does not possess the normal resistance against acid and pepsin. When this normal resistance in the stomach or in the duodenum is lacking it will also probably be absent in the esophagus. Therefore, peptic ulcer of the esophagus should be put on one line with peptic ulcer of stomach or duodenum.

A patient with an ulcer proximal or distal to the pylorus and also an ulcer in the esophagus is suffering from peptic digestion of the esophagus due to regurgitated stomach juice.

Another group of people suffers only from esophagitis. This has to be distinguished from peptic ulcer of the esophagus just as gastritis is differentiated from

peptic ulcer of the stomach. The cause of esophagitis remains unknown just as the cause of gastritis.

Under the influence of Dr. Wangensteen, I always advise subtotal resection of the stomach for patients with an ulcer of the lower esophagus, pain and difficulty in swallowing.

Most of the patients I have so advised have refused to follow my suggestion because they doubted, and their physicians doubted whether a multilating operation on the stomach could influence the esophagus. Most of these patients have done remarkably well after careful and expert dilatation of the esophagus.

Dr. O. H. Wangensteen:—Yesterday I had the opportunity of showing some of the slides on patients to which Colonel Lodmell referred. Some of you will recall the case of Harley Blackley, in that series, upon whom I performed a three-quarter gastric resection in March, 1948 for stenosing esophagitis of high grade. The stenotic area involved an area in the esophagus beginning about at the level of the bifurcation of the trachea, extending down to within about 7 cm. from the esophagogastric juncture. Eight years prior to the gastric resection performed by me, a gastrostomy had been performed at the Wisconsin General Hospital in Madison because Mr. Blackley could not swallow his saliva. For all of those eight years, the patient sustained himself by gastrostomy feedings. When I did the gastric resection in 1948, the gastrostomy was taken down simultaneously. This situation represents the farthest extension of resection to cure idiopathic esophagitis that I have tried. Because of the extreme narrowing of Blackley's esophagus, a few dilations were made prior to surgery, and had to be continued for six months after operation—up until October, 1949, since which time there has been no need for further dilatations. His esophagus now admits a No. 42 French dilator without any difficulty. Moreover, Mr. Blackley eats raw carrots, celery, steak and hard rolls without the slightest difficulty. His films now show a fairly normal esophagus. And when Mr. Blackley first came, it was only with difficulty that a No. 5 ureteral catheter could be passed down through the strictured esophagus, which catheter was fished out of the stomach through the gastrostomy opening—a circumstance that permitted us to dilate the esophagus gradually up to the calibre of a 19 French bougie before undertaking gastric resection.

The improvement of idiopathic esophagitis following gastric resection is usually dramatic. Blackley's case represents, however, a result beyond what one might have anticipated in view of the stricture as demonstrated in the x-ray film. (See reference below.)

My first experience with gastric resection for stenosing esophagitis was with Mr. Lyle Worden in September, 1939. He had a bleeding ulcer and I did an emergency gastric resection. In the four preceding years, Mr. Worden had been a regular attendant in our Out-Patient Clinic, where he had been dilated more

than a hundred times. Following operation, we dilated his esophagus twice, following which he refused further dilatations.

A second patient was seen in 1942. Esophagitis had been present over a relatively short time. However, the improvement following gastric resection was startling. It was only then that I appreciated for the first time how gastric resection had made continued dilatation of Mr. Worden's esophagus unnecessary. In 1944, I did a gastric resection for an idiopathic cork-screw-like stricture in the case of Mrs. Ella Kuehn—who as far as we knew had never had a peptic ulcer. Since the cases of Worden in 1939 and Van Dyke in 1942, I had been on the lookout for similar cases. All 3 of these patients were middle-aged. Both of the first two had duodenal ulcer. Mrs. Kuehn did not. Her stricture was about 10 cm. above the esophagogastric juncture. She was a very good surgical risk. In addition to anasarca from poor nutrition, she had a large polycystic liver, the presence of which had been established surgically elsewhere. It had been believed that a large abdominal tumor was responsible in part for her poor nutrition. In a sense, the proposal of gastric resection for this esophageal stricture was more of an "experiment" than in the case of Blackley, for we knew in his case at least that he had a peptic ulcer. However, by the time that Mrs. Kuehn presented herself for treatment with a history of many more than a hundred esophageal dilatations, my eyes had been opened by the improvement attending gastric resection in the first two cases. Mrs. Kuehn's case, however, was the first deliberate, premeditated attack upon an esophageal stricture of unknown origin, presumably unattended by a peptic ulcer. The pre- and late postoperative roentgenograms on these patients have been published recently (Transactions Am. Acad. of Ophthalmol. and Otolaryngol. July-August, 1953). They offer striking evidence that steps taken to reduce the capacity of the stomach to secrete HCl, accompanied by a satisfactory drainage operation, will cure an idiopathic esophageal stricture.

In part, clinicians have been slow to accept the idea of acid-peptic injury of the esophageal mucosa because pathologists like Kaufmann, Bell and others ascribed the postmortem findings in esophagitis to "postmortem digestion". However, Hogarth Pringle, as long ago as 1919, pointed out that, digestion of the esophagus could occur in patients during life and reported several such occurrences. Moreover, none of us appreciated the great sensitivity of the esophagus to acid-peptic juice. During the past 10 years, much effort has been devoted to this aspect of the problem in the Experimental Surgical Laboratories at the University of Minnesota. And in a number of earlier sessions of these meetings, I have reported upon some of those studies: how obstruction of the pylorus in the dog, when histamine is administered simultaneously, will result in severe erosive esophagitis; that perforation attends this occurrence; that the dripping of acid-peptic juice obtained from the involved gastric pouch of dogs, into the esophagus of a cat may result in perforation of the cat's esophagus in a surprisingly short period of time. In fact, even bile and pancreatic juice may cause erosive esophagitis, but not in the same degree as does acid-peptic juice.

It is not easy to produce stenosing esophagitis in dogs—a circumstance which comes about spontaneously in man, attending the regurgitation of acid-peptic juice into the esophagus. In the dog, hemorrhage and perforation occur far more readily.

With reference to "spontaneous perforation", which Colonel Lodmell mentioned, there has been warm debate in recent years in the meetings of the American Thoracic Society. It is generally held that "spontaneous perforation" of the esophagus is essentially a hydrostatic phenomenon. I have asserted in discussing the subject in meetings of the American Thoracic Society that, so-called "spontaneous perforation" of the esophagus is acid-peptic in nature. The occurrence of perforation attending obstruction of the pylorus in dogs when accompanied by the administration of histamine or following the dripping of acid-peptic juice into the esophagus of cats was not evidence enough to convince Mackler of Chicago or Sampson of Oakland. More recently, however, even more conclusive experimental evidence has been mustered which, I believe, clinches the point beyond any further discussion. When the acid-secreting area of the dog's stomach is excised in a preliminary operation, and the pylorus is obstructed subsequently, the administration of histamine fails to provoke esophagitis or perforation. Moreever, the performance of a cervical esophagostomy, which diverts the saliva, increases the incidence of severe esophagitis and perforation—a circumstance which lends further proof of the acid-peptic origin of these perforations (Proc. Soc. Exper. Biol. & Med. **88**:307, 1955).

There are those who would have us believe, I know, that stenosing esophagitis has origins other than the regurgitation of acid-peptic juice: that such esophagitis is essentially and primarily an inflammation which starts in the submucosal layers of the esophagus. It is a nice theory, but in the light of the compelling evidence of the importance of acid-peptic regurgitation, I would be inclined to record it as interesting testimony of an observer whose eyes failed to appreciate the importance of the more significant evidence.

The observation that gastric resection cures idiopathic stenosing esophagitis in man was the circumstance which alerted us to the great sensitivity of the esophagus to erosion of acid-peptic juice. Moreover, we have continued to treat such strictures in our Clinic by gastric resection—and the number of cases reported in 1949 (Surg., Gynec. & Obst. **88**:560) has been increased considerably. Moreover, whereas this procedure in Minneapolis appears to be attended by dramatic successes, the films of some of which cases you have seen today—in some areas, there appears to be far less enthusiasm for the method. I am here to present evidence—and not to give testimony as a proponent of any therapeutic expedient. Time, the ultimate arbitrer of all things, will resolve the issue. It is my feeling, however, that when more interested observers, like Dr. Palmer here, become fully aware of the devastating effects of acid-peptic juice upon the esophageal mucosa,

the serendipitous observation that gastric resection will cure idiopathic esopha-geal stricture will find wide acceptance as a therapeutic measure of real value.

Dr. Lodmell obviously, like many of us, is interested in the mechanism of regurgitation of gastric juice into the esophagus. More needs to be learned about this. Robins and Jankelson (J.A.M.A. **87**:1961, 1926) observed when patients were placed prone on the x-ray table during fluoroscopic examination, that regur-gitation of barium occurred in 4 per cent of instances. I have the feeling that when anyone of us tastes food, long hours after having ingested it, that gastric juice is being regurgitated into our esophagus. Heartburn is without question a manifestation of such regurgitation. This occurrence could, of course, be verified by the aspiration of acid from an inlying duodenal tube placed in the esophagus.

Let me say once more that gastric resection not only cures incipient cases of esophagitis, but also stenosing idiopathic esophagitis. When we have a satis-factory manner of producing stenosing esophagitis experimentally, resistance to the acceptance of regurgitation of acid-peptic juice as a cause of idiopathic esophagitis will disappear. I would remind you too that I am not advocating gastric resection for strictures of the esophagus resulting from the ingestion of corrosive poisons. There, esophageal resection accompanied by esophagogastric anastomosis is to be employed—I have done 3 such cases, which have been emi-nently successful. Professor Auguste, of Lille, France, writes me that he has performed gastric resection for corrosive esophagitis and that he obtained a satisfactory result. I am inclined to believe that the stricturing process in the esophageal wall, attending the ingestion of corrosive substances, extends further through the wall of the esophagus than it does in the strictures which result from acid-peptic regurgitation. This latter point could be verified in the experience of those who have resected the esophagus for idiopathic stenosing esophagitis.

THE ESOPHAGUS DURING PREGNANCY[*]

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The purpose of this report is to draw attention to the possibility of the development of serious esophageal disease associated with pregnancy. The confessions of surprise by our obstetrician colleagues, that such a complication of pregnancy even exists, testifies as to its apparent rarity. Recent experiences on the Gastroenterology Service of Walter Reed Army Hospital, and a search of the literature, however suggests that the otherwise normal pregnancy may be responsible for esophageal abnormalities more often than is realized.

Concurrent with enlargement of the uterus during pregnancy, there is an increase in intraabdominal pressure, which may result in elevation and abnormal rotation of the stomach and a disassociation of the normal relationship between the cardia and the diaphragm[22]. Hiatus hernia may result. Rigler and Eneboe[14] observed 25 instances of this among 195 women studied during their third trimester of pregnancy. They were unable to demonstrate any consistent correltation between symptoms and the presence of hiatus hernia in these patients, and concluded that it was, in itself, of little clinical importance during pregnancy. Since, however, the condition persisted after delivery in 3 out of the 10 patients re-examined by them, and there are reports of strangulation of hiatus herniae during labor[3,17], it cannot be dismissed as completely innocuous. It should be kept in mind that hiatus hernia is most likely to develop in older, multiparous women late in pregnancy[14].

Whether the anatomic shifts caused by the uterine enlargement result in the production of hiatus hernia or not, the efficacy of the cardiac closing mechanism is impaired. Regurgitation of gastric contents into the esophagus may then occur more freely than usual. Those same anatomic shifts also may cause delay in gastric emptying[22], thereby enhancing transcardial reflux. The stage is thus set for the development of "reflux" or "peptic" esophagitis. The frequent vomiting which so often accompanies pregnancy plays a variable role in the etiology of this type of esophagitis, since there is very little consistent correlation between the severity of vomiting and the development of that condition. Esophagitis and its complications, while more likely to occur during a pregnancy associated with

severe vomiting, can develop even in the absence of vomiting. This merely emphasizes the importance of gastroesophageal reflux due to the factors discussed above in the production of esophagitis (Fig. 1).

The "reflux" concept of the pathogenesis of erosive esophagitis[1,2,10,23] has not been universally accepted. Flood et al[5], using intubation and roentgenological technics were able to demonstrate regurgitation in only 50 per cent of the esophagoscopically proven cases of esophagitis studied by them. Palmer[12], after careful study of the histopathology of erosive esophagitis, concluded that the initial lesion occurs in the *lamina propria mucosae*, not at the surface of the mucosa as would be expected. If the "reflux" idea is correct, it is presumed that the acid-peptic factor of gastric juice plays an important role in the production of the injury
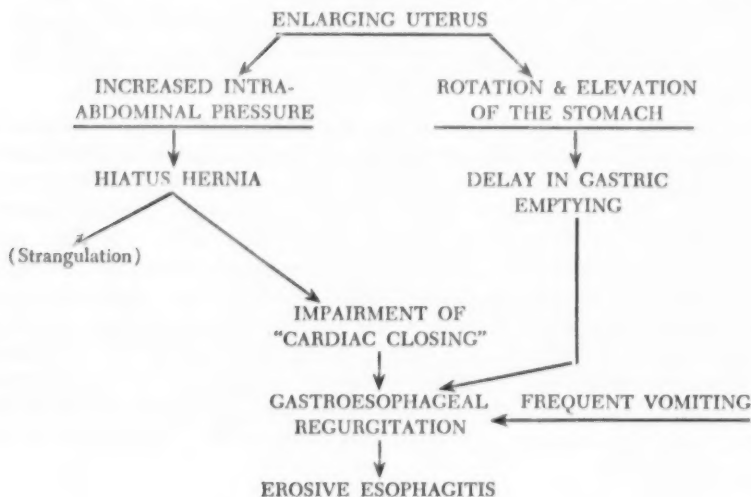
Fig. 1

done to the susceptible esophageal mucosa. The secretion of hydrochloric acid, essential for the activation of pepsinogen, however, is diminished and may be entirely supressed late in pregnancy[22]. Despite these objections to it, the "reflux" concept remains attractive because of its simplicity and ease of application. It is not the intent of this paper to enter the controversy over its validity.

Erosive esophagitis is the most common disease of the esophagus[11]. Whatever initiates the process, whether associated with pregnancy or not, the clinical course varies greatly. It may be limited to a simple, uncomplicated, often asymptomatic condition which clears spontaneously without sequelae upon termination of the underlying cause which permitted the excessive reflux across the esophagogastric junction. On the other hand, it may be a source of serious hemorrhage

during its acute stage, or it may result in stricture of the esophagus. Such a stricture is the result of fibrosis, and may occur quickly, or it may be delayed for varying lengths of time. This implies a transition of the esophagitis from an acute to a chronic stage. The clinical manifestations of this phase depend largely upon the degree of esophageal stenosis, and the presence or absence of a chronic "peptic" ulcer of the esophagus.

From 1921 through 1927, Vinson, at The Mayo Clinic, reported a total of 13 cases of esophageal stricture associated with pregnancies which had been complicated by severe vomiting[18,19,20]. This experience led him to estimate that 10 per cent of all benign strictures of the esophagus were the result of vomiting during pregnancy[21]. Until 1949, however, when Hara[6], published the account of a single similar case, and Rennie et al[13] reported 5 cases, no further mention of the combination is to be found. In 1952 Palmer[11] described another instance of esophageal stricture, and in the same year Hoover[7] reported 3 cases of esophagitis,

TABLE I

Author	Year	Diagnosis	Number
Vinson	1921-1927	Esophageal stricture	13
Hara	1949	Esophageal stricture	1
Rennie	1949	Esophageal stricture	5
Palmer	1952	Esophageal stricture	1
Hoover	1952	Hemorrhagic esophagitis	1
		Esophageal stricture	2
Johnstone	1952	Esophageal ulcer	2
Schmidt	1954	Esophageal ulcer	2

all believed to be due to vomiting during pregnancy. One of Hoover's patients developed hematemesis and the other two stricture of the esophagus. Also in 1952, Johnstone[8] presented 2 cases of "peptic" ulcer of the esophagus with stricture associated with excessive vomiting during pregnancy. Recently, Schmidt[15], in a review of 170 cases of regurgitant ulceration at the esophagogastric junction, cited two as being due to vomiting during pregnancy; one of these developed stricture of the esophagus (Table I).

During the period from June, 1952 until January, 1954, seven cases were observed on the Gastroenterology Service of Walter Reed Army Hospital in which the association of pregnancy with esophagitis or its complications was reasonably clear. These cases were characterized by considerable variability in clinical course and outcome.

1. *Simple erosive esophagitis* manifested by dysphagia and substernal fullness followed a 3-day period of severe vomiting just before delivery in a 41-year

old white multiparous woman. She was asymptomatic after one week. Treatment with bismuth subcarbonate, (administered orally for its protective coating action) and elevation of the head of her bed to reduce transcardial reflux were probably of less benefit than termination of pregnancy. She has remained completely well for over a year. This case represents the mildest form of the disease, which may be entirely asymptomatic and, therefore, frequently is overlooked.

2. *Hemorrhagic esophagitis* characterized by repeated episodes of hematemesis of moderate degree and associated with dysphagia and substernal fullness began during the fifth month of gestation of a 24-year old secundagravida. Vomiting had been minimal. Barium swallow x-ray study was normal, but esophagoscopy revealed severe erosive esophagitis, with diffuse bleeding from the mucosa. There was marked reflux from the stomach into the esophagus. No further hematemesis occurred after institution of treatment similar to that employed in Case 1, although slight dysphagia persisted. It is of importance that only minimal vomiting occurred in this case. Follow-up is not complete in this patient.

3. *Hematemesis of severe degree occurred and stricture developed rapidly* in a 29-year old white primagravida who experienced severe vomiting for 14 days at the end of her pregnancy. A nasogastric tube employed for feeding purposes for 5 days surely contributed to the production of the hemorrhagic esophagitis and stricture, but the importance of regurgitation of gastric contents through the incompetent cardia, and the excessive vomiting must not be minimized. This patient had experienced severe heartburn earlier in her pregnancy; its importance as a symptom of esophagitis at that time is not clear (Fig. 2).

4. *Stricture in the form of a web* in the proximal esophagus was found in a 30-year old secundagravida *who did not vomit* at all during her second pregnancy. Dysphagia began during the seventh month of that pregnancy, and persisted with increasing severity for 2 years, at which time transient total obstruction occurred due to impaction of food. Esophagoscopy at that time revealed the web. A web may be congenital, but it is usually the result of inflammation, and represents a special type of stricture. Reflux of gastric contents has been demonstrated to rise, in some individuals, to as high as the cricopharynx, and it is postulated to be the cause of the esophagitis which resulted in the formation of the web in this case.

5. *Stricture of the esophagus* was observed in a 72-year old white woman, which had its beginning at the age of 23 years *immediately* following termination of her fourth pregnancy, which had been complicated by *hyperemesis gravidarum*. Periodic dilatations have been necessary for nearly 50 years in order to maintain a patent esophageal lumen (Fig. 3).

6. *Delayed stricture* occurred in 2 of our cases. One, a 36-year old white primagravida, experienced severe vomiting during the final week of her preg-

nancy. There were no gastrointestinal symptoms following delivery until the onset
of dysphagia one month later. This progressed until complete obstruction of the
esophagus occurred within three weeks. At present, two years later, she still
requires periodic esophageal dilatations (Fig. 4).

7. A less credible case is that of a 40-year old white primagravida who experi-
enced severe vomiting throughout her only pregnancy at the age of 27. Esophageal
stenosis and possible ulcer of the mucosa became clinically evident 3 years after
delivery, and she has required periodic treatment ever since. A recent esophagram
shows narrowing in the distal esophagus, and what have been interpreted as

Fig. 2   Fig. 3

Fig. 2—Esophagram from Case 3, showing irregularity of the mucosa and narrowing of the
       esophageal lumen.
Fig. 3—Barium swallow x-ray showing narrowing of the esophageal lumen. Case 5.

two pseudodiverticula. Esophagoscopy confirms the slight narrowing, and shows
evidence of chronic esophagitis. There is no evidence of hiatus hernia nor of
shortening of the esophagus to account for the esophageal abnormalities. Stother[16]
in reviewing his experiences with esophageal stricture due to corrosive agents
noted that stricture did not occur in some instances for as long as 50 years after
the initial trauma. Why could not such a delay have occurred in our case, the
damage being initiated by the gastroesophageal reflux and excessive vomiting
during pregnancy three years earlier, rather than by ingested corrosive material
(Fig. 5)?

Of the 34 cases made available by combining those just presented with the
ones reviewed from the literature, only one had simple, uncomplicated erosive

esophagitis. It is apparent that two serious complications of erosive esophagitis, hemorrhage and stricture, may develop in an unpredictable manner, and that frequently the early, simple stage is overlooked because symptoms, when present, may be variable and unreliable. The absence of symptoms does not rule out the presence of esophagitis. Assuming that erosive esophagitis preceded every case of serious esophageal disease herein described, analysis of these cases was undertaken in an attempt to glean further information about the primary lesion. No magical treatment of esophagitis is known, but it is reasonable to anticipate that early diagnosis and treatment with the means at hand might at least slow the progress of the development of complications. The return of the uterus to
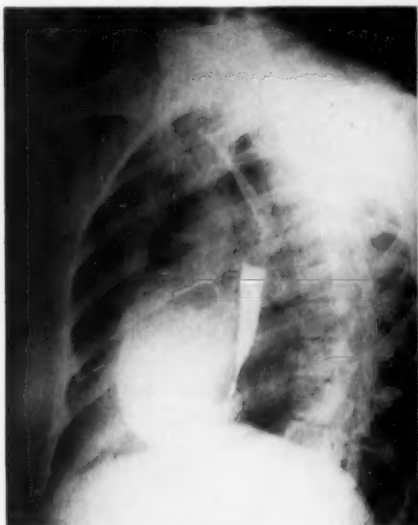
Fig. 4   Fig. 5

Fig. 4—Esophagram from Case 6. There is complete stricture of the esophagus.
Fig. 5—Esophagram from Case 7 showing stenosis in the distal esophagus. The irregularities within the narrowed area were interpreted as pseudodiverticula.

normal size at the completion of pregnancy is probably the most effective therapeutic occurrence, but even this does not always result in a remission.

The average age of these patients was 34 years. All were white. The majority were multiparous, although 6 of them developed esophageal disease during their first pregnancy. Vomiting occurred in 19 of the 27 patients in whom that information is available, but in only 9 of these was it of sufficient severity to warrant the diagnosis of *hyperemesis gravidarum*. In the other 12 the vomiting was no more severe then that experienced by many women, and endured by them as an almost natural part of pregnancy. Of great interest are the remaining 6 in whom no vomiting occurred. It must be assumed that gastroesophageal reflux through a incompetent cardia was the cause of the esophagitis in those cases.

Winkelstein[23] has observed that dysphagia is the only reliable symptom of esophagitis. In a review of 20 cases of "peptic" esophagitis he concluded that that diagnosis could not be supported by complaints of heartburn, regurgitation, and substernal pain, in combination or singly, in the absence of dysphagia. Heartburn, which is a common complaint during pregnancy, is known to be associated with hiatus hernia and esophagitis, but it has never been consistently correlated with any organic lesion, and must be considered thoroughly unreliable. Regurgitation, considered by some never to be asymptomatic[16], is actually not abnormal in all instances, and does not always produce esophagitis. The esophageal mucosa has been demonstrated to be more susceptible to the digestive action of this refluxed gastric juice than either stomach or duodenal mucosae[4,9]. Nevertheless, it posesses some inherent protective mechanism, the effectiveness of which obviously varies considerably between individuals and probably in the same individual at different times. Substernal pain, unless initiated by swallowing, (and then it is dysphagia) cannot reliably be attributed to esophageal disease. The inconstancies of referred pain mechanisms precludes any such pinpoint localization of its source.

Unfortunately, dysphagia seemed to occur almost simultaneously with the appearance of the complication in many of our cases, and it was not a completely reliable warning signal. A truly accurate evaluation of the symptoms experienced during pregnancy with regard to their possible relationship to esophagitis demands esophagoscopic correlation. It is obviously impractical to carry out such a procedure on all pregnant women who complain of heartburn, regurgitation or substernal fullness. X-ray is of limited value in the early case. Esophageal spasm and mucosal erosions are direct evidences of esophagitis, but since they are only rarely present in the early stage, their absence cannot be interpreted as evidence to rule out esophagitis. Transcardiac regurgitation, with or without hiatus hernia, is only indirect evidence of esophagitis, and may not be associated with that condition at all.

Dysphagia, occurring during pregnancy, should be viewed with at least mild alarm, and interpreted to indicate the presence of esophagitis. Severe vomiting should alert us that esophagitis may develop, but, like heartburn, regurgitation, and substernal pain without dysphagia, this is not necessarily associated with esophageal inflammation. Treatment with bismuth subcarbonate and elevation of the head of the patient's bed is certainly indicated in the pregnant woman who complains of dysphagia. Actually, it probably should be instituted in any instance where there is doubt as to the exact significance of the other less reliable symptoms. The bismuth subcarbonate should be administered orally in its dry, powder form, for its protective, coating action on the esophageal mucosa. The head of the patient's bed should be elevated at least 8 inches to be most effective in reducing regurgitation. These measures may have their chief value in the slight ability they possess to ameliorate the symptoms of heartburn, regurgitation and substernal pain.

Little can be said regarding the asymptomatic cases of esophagitis, except to re-emphasize the need to be aware that such a condition may arise as a consequence of the anatomic changes inherently associated with pregnancy, even in the absence of vomiting.

SUMMARY

Erosive esophagitis may occur in association with the otherwise normal pregnancy. It is most likely to occur in multiparous white women, over the age of 30 years, who vomit excessively during pregnancy, and may progress insidiously to develop serious complications such as hemorrhage and esophageal stricture. It must not be forgotten, however, that the same process may develop and progress in younger, primiparous patients, even in the absence of vomiting. Anatomic changes inherently associated with pregnancy are probably responsible for the gastroesophageal regurgitation which produces erosive esophagitis in such instances. Erosive esophagitis, in its simple, uncomplicated stage may be entirely asymptomatic. Dysphagia is the only reliable symptom of esophagitis, while heartburn, regurgitation, and substernal pain, in combination or singly, are not consistently associated with esophageal disease. Expectant treatment, with bismuth subcarbonate and elevation of the head of the patient's bed is advisable in any case in which there is doubt as to the exact significance of these symptoms. Of primary importance is the need for awareness that erosive esophagitis and its complications may be associated with pregnancy.

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DISCUSSION

Dr. I. Snapper:—This interesting paper clearly indicates that the etiology of many cases of unexplained esophageal lesions in women is closely connected with hyperemesis during pregnancy.

The fact that it is not quite established is whether the majority of pregnant women have hyperacidity should not be a reason to doubt the close etiologic connection which evidently exists between pregnancy and esophagitis.

It is certain that acid and pepsin play an important role for the causation of ulcers of the stomach and of the duodenum. But this does not prove that increase of acid and pepsin constitutes the *only* cause for the ulcer, or even the preponderant cause of the ulcer. The preponderant place of subtotal gastrectomy in the treatment of ulcers resistant to conservative therapy has perhaps had too great an influence upon our thinking.

We know that an ulcer of the stomach may be found in the presence of hypo- or even achlorhydria; it may even grow in the absence of hydrochloric acid. Thus the absence of hyperacidity during pregnancy should be no reason to doubt the frequency of esophagitis in pregnancy. During pregnancy signs of a moderate hyperfunction of the eosinophilic cells of the pituitary develop. The nose becomes larger, the lips thicker and laryngoscopic examination reveals thickening of the mucous membrane of the vocal cords. Maybe the tissues of the esophagus also change whereby this organ can become more susceptible to ulceration even if acid and pepsin are secreted in normal quantities. An observation is often correct although the explanation may come very much later!

President's Message

This will be the last time it will be my privilege to address you before our annual meeting in Chicago. In the interests of co-ordination, I should like to remind the Committee Chairmen that they must meet with their Committees some time before the meeting of the Board of Trustees on Sunday morning, October 23rd. That means you should arrive sometime Saturday.

The Board of Trustees will need your reports and recommendations in time for their final meeting of the year, Sunday morning.

The annual meeting of the College will be held Sunday afternoon, and all Fellows in good standing are urged to arrive early, and be present to vote. A buffet supper will be at 6 o'clock and the Convocation ceremony will follow, about 7:30 o'clock. Consequently, it becomes necessary for the Candidate and the entire membership to assemble early Sunday, in order to get acquainted, and to become a part of that important event. These are a few innovations to be tried out this year, so that we will not be pressed for time later in the week. It is hoped you will make your reservations now, and take advantage of the additional week-end time for these important parts of the agenda. The Convocation is an impressive ceremony to which you may invite your friends, and let's try to make creditable tradition.

Dr. Kirchner's Convention Program is the best we have ever had, and credit goes to him, and his Committee, for its perfection.

The Postgraduate Course Program, under Dr. Wirts, is exceptionally well thought out and promises to be more than usually popular. I discussed the advance registrations with our Executive Secretary, Dan Weiss, last week, and it appears that the attendance will far exceed previous years. This is most encouraging, and repays the effort.

Drs. Wangensteen and Snapper are well known for their ability as coordinators. We urge our own men to remain over, and take advantage of this important opportunity to improve themselves.

Women's events are taking shape rapidly, and will soon be perfected. Your help is solicited and, particularly, in getting the men to know each other.

I pledge my whole-hearted support with you to maintain our College at the very highest level. Will see you in Chicago early, about Saturday, the 22nd.

Lynn. A. Ferguson

EDITORIAL

The Simple Water Tap Enema — A Warning

Although physicians in general and gastroenterologists in particular prescribe a plain water tap or medicated enema in constipation, previous to sigmoidoscopy, cholecystography or barium enema, little do they know the possible dangers of these enemas in some individuals.

In the editor's column in the *Journal of Pediatrics,* December, 1954, attention is called to the danger of these cleansing enemas even in normal patients, especially when plain water is used. A small enema may have little or no effect upon the electrolyte balance of the patient, however, a large quantity of hypotonic solution may cause an increased blood volume and secondary water intoxication, pulmonary edema and death.

Richards and Hiatt called attention to the untoward effects of enemas in congenital megacolon. They said they were due to the increased hydrostatic pressure in the oversized colon with the increased absorptive surface, causing a rapid fall in the plasma concentration of the blood sodium chloride and thus inducing a hynatremic shock. They advocate enemas of isotonic saline solution in patients with megacolon, although in renal and cardiac patients even isotonic enemas should be administered guardedly. In the latter conditions, a 7 per cent gelatin enema is preferable and if intoxication does occur, intravenous 2 per cent sodium chloride should be the routine procedure.

Prouty found that the electrolyte balance in infants and children is easily upset because half of the extracellular fluid is in the form of daily water intake and output; whereas the daily intake and output of water in an adult is only one-seventh of the extracellular fluid. Helwig and his co-workers reported the death of a patient who developed cerebral edema produced by disturbance in the normal isotonicity of the blood by proctolysis after a simple cholecystectomy. They found that the patient absorbed nine liters of water in 30 hours.

The symptoms of water intoxication are weakness, vomiting, sweating, pallor, clammy skin, cough, syncope and listlessness. Treatment should not be delayed. Intravenous infusions of 5 per cent dextrose, isotonic saline solution and parasympathonimetic agents, such as neostigamine should be given at the first sign of untoward symptoms.

As a further precaution in preventing water intoxication, it is advocated that isotonic sodium chloride solution be employed for all enemas including the barium enema.

Samuel Weiss, M.D., F.A.C.G.

NEWS NOTES

SECOND ANNUAL CONVENTION

The Second Annual Convention of the American College of Gastroenterology will be held at The Shoreland in Chicago, Ill., on 24, 25, 26 October 1955.

Copies of the excellent program for the Convention are being mailed separately to the membership. Additional copies are available from the headquarters office, 33 West 60th Street, New York 23, N. Y. They will also be available at the registration desk on the Convention floor.

There will be many instructive and informative papers presented by members of the College, as well as invited guests. Dr. Arthur A. Kirchner and his committee have spared nothing in their efforts to bring the foremost authorities to the Convention.

Several changes in the times of the various parts of the program have been made and the Trustees, Governors, Members of Committees and Fellows, are urged to check the program and arrange their arrival date accordingly.

LADIES AUXILIARY PROGRAM

Under the Presidency of Mrs. Lynn A. Ferguson and the Program Chairmanship of Mrs. Joseph Shaiken, the Ladies Auxiliary has prepared a most interesting and exciting round of events during the Convention days and immediately following the Convention, during the Postgraduate Course.

Complete details of all the events are being sent by mail to the ladies.

The program follows:

Sunday, 23 October 1955

Registration opens at 1:00 P.M. A badge is necessary in order to visit the exhibits.

Business meeting of the Auxiliary and election of officers at 3:00 P.M.

Buffet supper 6:00 P.M.

Convocation Ceremony 7:30 P.M.

Monday, 24 October 1955

Social get together and coffee hour at The Shoreland at 10:00 A.M.

Luncheon at the Museum of Science and Industry (optional) at 12:30 P.M.

Tour of the Museum of Science and Industry 1:45 P.M.

Tuesday, 25 October 1955

Two hour sightseeing tour of Chicago including the Loop area with buses leaving the hotel at 9:00 A.M.

Tour of the Merchandise Mart and luncheon at the famous Henrici's in the Mart 11:00 A.M.

Cocktail party and Annual Banquet of the Association at The Shoreland at 7:00 P.M. This annual event is *informal*. Dancing and entertainment will follow the banquet.

Reservations must be made in advance.

Wednesday, 26 October 1955

Radio program at WGN 9:30 A.M. Free tickets available at the registration desk.

Luncheon and fashion show at the fabulous Marshall Fields, 12:45 P.M.

Thursday, 27 October 1955

Tour of the Swift & Co. plant, with luncheon as their guests, starting at 10.00 A.M.

Friday, 28 October 1955

Tour of the Chicago Tribune newspaper printing plant at 2:00 P.M.

REGISTRATION

Registration for the Convention will take place on the mezzanine floor at The Shoreland commencing at 2:00 P.M., on *Sunday, 23 October 1955*. Those attending are requested to register and receive their identification badges as no one will be admitted to the exhibits or sessions without a badge.

BOARD OF TRUSTEES

The Annual Meeting of the Board of Trustees will be held at The Shoreland in Chicago, Ill., at 9:00 A.M., on Sunday, 23 October 1955. A luncheon for the Trustees will follow the meeting.

ANNUAL MEETING OF THE AMERICAN COLLEGE OF GASTROENTEROLOGY

The Annual Meeting of the American College of Gastroenterology will be held at The Shoreland in Chicago, Ill., on Sunday afternoon, 23 October 1955 at 3:00 P.M. Election of Officers, Trustees and Members of the Board of Governors will be held at that time.

Fellows of the College are requested to attend and participate in the business session.

BUFFET SUPPER

A buffet supper for those attending the Convention on Sunday, 23 October 1955 will take place at The Shoreland in Chicago, Ill., between the annual meetings of the College and the Ladies Auxiliary at 3:00 P.M. and the convocation ceremony at 7:30 P.M.

The supper will be held in the Louis XVI Ballroom at 6:00 P.M. and tickets are available at the registration desk.

CONVOCATION CEREMONY

The Convocation Ceremony, at which certificates will be presented to newly elected and advanced Associate Fellows, Fellows and Honorary Fellows, will this year be held on Sunday evening, 23 October 1955 at The Shoreland in Chicago, Ill.

An interesting and impressive ceremony has been planned. Members, their families, guests and friends are invited to attend.

COFFEE HOUR

Following last year's successful coffee hour, coffee and sweet rolls will again be served in the exhibit area on Monday, Tuesday and Wednesday mornings from 8:30 A.M. to 9:00 A.M. Again this year this will be furnished with the compliments of the College and those attending the Convention are invited to join us.

SCIENTIFIC EXHIBITS

The Scientific Exhibit Committee under the chairmanship of Dr. Michael W. Shutkin, have assembled several outstanding scientific exhibits which will be on display in the exhibit area from Monday through Thursday.

The committee will select the best exhibits, for which prizes will be awarded.

BURTON, PARSONS & CO. LUNCHEON

Continuing the custom inaugurated last year, Burton, Parsons & Co. of Washington, D.C., will sponsor an annual luncheon for those attending the sessions.

The luncheon will be held on Monday, 24 October 1955 and the speaker will be Dr. Martin E. Rehfuss of Philadelphia.

Tickets of admission may be obtained at the registration desk on the Convention floor.

MEETING OF THE BOARD OF GOVERNORS

The Annual Meeting of the Board of Governors will be held at The Shoreland in Chicago, Ill., on Tuesday, 25 October 1955 at 4:00 P.M.

The election of a chairman and appointment of committees will take place.

ANNUAL BANQUET

The annual banquet of the College will be held at The Shoreland in Chicago, Ill., on Tuesday evening, 25 October 1955 at 7:00 P.M. The banquet will be preceded by cocktails and will be followed by dancing and entertainment.

There will be several prominent invited guests but there will be no formal speakers.

Dr. James T. Nix of New Orleans, La., the new incoming President, will be installed at that time.

Tickets at $7.50 per person will be available at the registration desk and this is an *informal* affair.

All reservations must be made by 10:00 A.M., Tuesday, 25 October 1955.

Course in Postgraduate Gastroenterology

The annual Course in Postgraduate Gastroenterology of the American College of Gastroenterology will be held at The Shoreland in Chicago, Ill., on 27, 28, 29 October 1955.

The excellent program has been arranged by Dr. C. Wilmer Wirts and his committee and we will again have our two co-ordinators, Dr. Owen H. Wangensteen, Professor and Chairman of the Department of Surgery, University of Minnesota Medical School and Dr. I. Snapper, Director of Medical Education, Beth-El Hospital, Brooklyn, N. Y., with us.

The outstanding faculty assisting Drs. Wangensteen and Snapper has been chosen from the various medical schools throughout the country.

Admission to the Course sessions is limited to those who hold matriculation cards indicating that they have paid their fee for the Course.

Nominating Committee Report

The Nominating Committee of the American College of Gastroenterology, consisting of Dr. Sigurd W. Johnsen, Passaic, N. J., Chairman; Dr. James T. Nix, New Orleans, La.; Dr. Edward J. Krol, Chicago, Ill.; Dr. Lester M. Morrison, Los Angeles, Calif. and Dr. Michael W. Shutkin, Milwaukee, Wisc., has submitted the following slate of candidates to be voted upon at the Annual Meeting of the College in October:

Officers

President-Elect . . . . Arthur A. Kirchner, M.D., Los Angeles, Calif.
1st Vice-President . . . . C. Wilmer Wirts, M.D., Philadelphia, Pa.
2nd Vice-President . . . . Frank James Borrelli, M.D., New York, N. Y.
3rd Vice-President . . . . Joseph Shaiken, M.D., Milwaukee, Wisc.
4th Vice-President . . . . Henry Baker, M.D., Boston, Mass.
Secretary . . . . Theodore S. Heineken, M.D., Bloomfield, N. J.

Board of Trustees

For 3 years: Edward J. Krol, M.D., Chicago, Ill.
                                    Harry M. Eberhard, M.D., Philadelphia, Pa.
                                    William C. Jacobson, M.D., New York, N. Y.
                                    Donald C. Collins, M.D., Hollywood, Calif.
                                    Samuel S. Berger, M.D., Cleveland, Ohio
For 1 year: To fill his own unexpired term, ending in 1956—
                                    Fred H. Voss, M.D., Phoenicia, N. Y.

Program

AMERICAN COLLEGE OF GASTROENTEROLOGY

SECOND ANNUAL CONVENTION
24, 25, 26 OCTOBER 1955
and
COURSE IN POSTGRADUATE GASTROENTEROLOGY
27, 28, 29 OCTOBER 1955

THE SHORELAND
5454 South Shore Drive, Chicago, Ill.

Members of the medical profession are cordially invited to attend the convention sessions.

Attendance at the Postgraduate Course is limited to those who have paid the matriculation fee.

REGISTRATION—All members and guests should register. Identification badges for admittance to meetings will be given to those who register. These should be worn at all times during the session. Registration will take place at the registration desk on the convention floor.

LADIES REGISTRATION—At the registration desk on the Convention Floor. Registration facilities will be open at 8:30 each morning. Information concerning the various activities and events will be available there.

MEETINGS are held on local time and will begin promptly at the time specified.

COURSE IN POSTGRADUATE GASTROENTEROLOGY—Admittance only upon presentation of official matriculation card.

SCIENTIFIC EXHIBITS—Will be in the Exhibit Hall and will be open Monday, Tuesday, and Wednesday 8:30 a.m. to 5:00 p.m., Thursday from 8:30 a.m. to 2:00 p.m.

TECHNICAL EXHIBITS under the direction of Mr. Steven K. Herlitz, Exhibit Manager, will be open Monday, Tuesday, and Wednesday from 8:30 a.m. to 5:00 p.m., Thursday from 8:30 a.m. to 2:00 p.m.

Those attending the Convention are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with the many new products and new equipment on display.

VISIT THE EXHIBITS

PROGRAM

SECOND ANNUAL CONVENTION
AMERICAN COLLEGE OF GASTROENTEROLOGY

SCIENTIFIC SESSIONS
24, 25, 26 October 1955

and

COURSE IN POSTGRADUATE GASTROENTEROLOGY
27, 28, 29 October 1955

THE SHORELAND

5454 South Shore Drive

Chicago, Ill.

SPEAKERS AND OFFICERS OF INSTRUCTION

ADOMAVICIUS, JONAS, M.D., Chicago, Ill. Cook County Hospital. *(p. 21)*.

BALFOUR, DONALD C., Jr., M.S., M.D., Los Angeles, Calif. Assistant Clinical Professor of Medicine, University of Southern California, School of Medicine. *(pp. 11, 14)*.

BARBORKA, C. J., M.D., Chicago, Ill. Associate Professor of Medicine, Northwestern University Medical School; Attending Physician, Passavant Memorial Hospital; Senior Consultant in Gastroenterology, Veterans Administration Research Hospital. *(p. 31)*.

BEASLEY, L. K., A.B., M.D., St. Louis, Mo. Chairman, Medical Department, Evangelical Hospital. *(p. 15)*.

BEMAN, FLOYD, M., A.B., M.D., Columbus, Ohio. Associate Professor, Department of Medicine, Ohio State University, Ohio State University Hospital. *(p. 23)*.

BERGER, SAMUEL S., M.D., F.A.C.G., Cleveland, Ohio. Consultant, Mt. Sinai Hospital. *(p. 17)*.

BERRY, LEONIDAS H., M.D., F.A.C.G., Chicago, Ill. Professor of Gastroenterology, Cook County Postgraduate School of Medicine; Assistant Clinical Professor, University of Illinois Medical School. *(p. 21)*.

BLALOCK, JOHN, B.A., M.D., F.A.C.S., New Orleans, La. Instructor in Surgery, Tulane University, School of Medicine; Ochsner Clinic and Ochsner Foundation Hospital; Visiting Surgeon, Charity Hospital. *(p. 25)*.

BOHROD, MILTON G., M.D., Rochester, N. Y. Pathologist and Director of Laboratories, The Rochester General Hospital. *(p. 10)*.

BORNEMEIER, W. C., B.A., M.D., F.A.C.S., F.I.C.S., F.A.C.G., Chicago, Ill. Attending Surgeon, Illinois Masonic Hospital; Consulting Surgeon, Resurrection and Alexian Brothers Hospitals. *(p. 24)*.

BROWN, DAVID B., M.D., Columbus, Ohio. Resident, Gastroenterology Division, Ohio State University Hospital. *(p. 23)*.

CANAHUATI, SHIBLI M., M.D., Chicago, Ill. Resident in Internal Medicine. *(p. 13)*.

CAROLI, JACQUES, M.D., F.A.C.G., Paris, France. Chief of Medicine, Hospital, St. Antoine. *(p. 11)*.

CARROLL, WALTER W., M.S., M.D., F.A.C.S., Chicago, Ill. Assistant Professor of Surgery, Northwestern University Medical School; Department of Surgery, Passavant Memorial Hospital. *(p. 13)*.

CHAIKIN, NATHAN W., B.S., M.D., F.A.C.P., F.A.C.G., New York, N. Y. Associate Professor of Clinical Medicine, New York Medical College. *(p. 18)*.

VISIT THE EXHIBITS

CHILD, CHARLES G., III., A.B., M.D., Boston, Mass. Professor of Surgery, Tufts College Medical School; Director of the First (Tufts) Surgical Service, Boston City Hospital. (p. 10).

CHRISTIAN, EDWARD R., A.B., M.D., New Orleans, La. Instructor in Medicine, Tulane University, School of Medicine; Assistant Chief of Medicine, Veterans Administration Hospital. (p. 11).

COHN, ISIDORE, D.Sc., M.D., F.A.C.S., New Orleans, La. Consulting Surgeon, Tuoro Infirmary, Charity Hospital. (p. 9).

COLE, T. J., M.D., Chicago, Ill. Provident Hospital. (p. 21).

COLEMAN, JOHN M., B.S., M.D., Chicago, Ill. Instructor in Clinical Medicine, Stritch School of Medicine, Loyola University; Mercy Hospital. (p. 14).

CREEK, DALE W., A.B., M.D., Santa Barbara, Calif. Santa Barbara Cottage, Santa arbara General and St. Francis Hospitals. (p. 12).

CURRERI, ANTHONY R., B.A., M.D., F.A.C.S., Madison, Wisc. Professor of Surgery, University of Wisconsin, School of Medicine. (p. 26).

DALESSANDRO, WILLIAM, M.D., Chicago, Ill. Associate Staff, Augustana Hospital. (p. 13).

DeFEO, H. F., B.S., M.S., M.D., F.A.C.P., F.A.C.S., F.A.C.G., Chicago, Ill, Associate Clinical Professor of Medicine, Stritch School of Medicine, Loyola University. (p. 22).

DeLOR, C. JOSEPH, M.D., F.A.C.G., Columbus, Ohio. Associate Professor, Department of Medicine, Ohio State University. (p. 23).

EICHHORN, RALPH D., M.D., Houston, Texas. Assistant Professor of Clinical Medicine, Baylor University, College of Medicine. (p. 10).

FARFEL, BERNARD, B.S., M.D., Houston, Texas. Assistant Professor of Clinical Medicine, Baylor University, College of Medicine. (p. 10).

FEDER, ISIDORE A., M.D., F.A.C.P., F.A.C.G., Brooklyn, N. Y. Adjunct Professor of Gastroenterology, New York Polyclinic Medical School and Hospital; Attending Physician, Beth-El Hospital. (p. 18).

FIERST, SIDNEY M., B.S., M.D., M.S., F.A.C.P., F.A.C.G. Brooklyn, N. Y., Clinical Assistant Professor of Medicine, State University Medical Center, New York City; Physician-in-Charge, Gastroenterology, Kings County Hospital (University Division) and Maimonides Hospital; Attending Gastroenterologist, Veterans Administration Hospital. (p. 17).

FISHER, MICHAEL, M.D., Brooklyn, N. Y. Proctologist, Maimonides Hospital; Associate Surgeon, Coney Island Hospital. (p. 17).

FRITZ, I. B., Ph.D., D.D.S., Chicago, Ill. Acting Assistant Director, Department of Metabolic and Endocrine Research, Michael Reese Hospital. (p. 25).

FRYE, WILLIAM W., B.S., M.S., Ph.D., M.D., New Orleans, La. Dean, Louisiana State University, School of Medicine, Senior Visiting Physician, Charity Hospital. (p. 12).

GECHMAN, ELIAS, M.D., Brooklyn, N. Y. Fellow in Medicine, Beth-El Hospital. (p. 18).

GIANTURCO, CESARE, M.D., M.S., Urbana, Ill. Roentgenologist, Carle Hospital Clinic. (p. 15).

GRANET, EMIL, A.B., M.D., F.A.C.G., New York, N. Y. Lecturer in Graduate Medicine, Columbia University; Associate Surgeon (Proctology), French Hospital. (p. 15).

GRAY, SEYMOUR J., M.D., Ph.D., F.A.C.P., Boston, Mass. Assistant Professor of Medicine, Harvard Medical School, Senior Associate in Medicine, Peter Bent Brigham Hospital. (p. 9).

GUYNN, VERNON L., B.S., M.D., Chicago, Ill. Instructor in Anatomy, University of Illinois, College of Medicine; dispensary Staff, St. Luke's Hospital. (p. 16).

HALPERN, SEYMOUR LIONEL, A.B., M.D., F.A.C.G., New York, N. Y. New York Medical College, Flower and Fifth Avenue Hospitals; Physician-in-Chief, Nutrition Clinic, Bureau of Nutrition, New York City Department of Health; Metropolitan, Bird S. Coler Memorial and Beth Israel Hospitals; Food and Nutrition Section, Health and Welfare Council, New York City. (p. 19).

HWANG, KAO, M.D., Ph.D., Chicago, Ill. Research Associate, Department of Clinical Science, University of Illinois; Senior Pharmacologist, Research Division, Abbott Laboratories. (p. 21).

VISIT THE EXHIBITS

JEFFERSON, N. C., M.D., Chicago, Ill. Provident Hospital; Gastrointestinal Research Department, Michael Reese Hospital. *(p. 25)*.

KALB, S. WILLIAM, M.D., F.A.C.G., Newark, N. J. Beth Israel Hospital; Chairman, New Jersey Nutrition Council. *(p. 17)*.

KAPLAN, MURREL H. A.B., M.D., F.A.C.G., New Orleans, La. Senior, Department of Gastroenterology, Touro Infirmary. *(p. 12)*.

KAPLAN, ROBERT S., M.D., Los Angeles, Calif. Senior Surgical Resident, Cedars of Lebanon Hospital. *(p. 20)*.

KEANE, JOHN F., A.B., M.D., F.I.C.S., F.A.C.G., Boston, Mass. Boston Dispensary, New England Medical Center; Proctologist, Veterans Administration, Boston Regional Office; Surgeon, St. Elizabeth's, Newton-Wellesley, Sancta Maria Hospitals. *(p. 17)*.

KIRSNER, JOSEPH B., M.D., Ph.D., Chicago, Ill. Professor of Medicine, University of Chicago, School of Medicine; Attending Physician, Albert Merritt Billings Hospital. *(p. 24)*.

KONIGSBERG, MAX, M.D., New York, N. Y. Associate Physician, Metropolitan Hospital. *(p. 18)*.

KUZMA, J. F., B.S., M.D., M.S., Milwaukee, Wisc. Professor and Director, Department of Pathology, Marquette University, School of Medicine; Consultant in Pathology, Milwaukee County Hospital and Veterans Administration Hospital. *(p. 22)*.

LARY, BANNING G., B.S., M.S., M.D., Miami, Fla. Formerly Instructor in Surgery, University of Illinois, College of Medicine; Fellow, St. Luke's Hospital. *(p. 16)*.

LAUFMAN, HAROLD, Ph.D., M.D., Chicago, Ill. Associate Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Passavant Memorial Hospital. *(p. 22)*.

LEVIN, IRVING A., B.S., M.D., F.A.C.S., F.A.C.G., New Orleans, La. Surgery, Louisiana State University, School of Medicine; Visiting Surgeon, Charity Hospital; Hotel Dieu, Sara Mayo and Touro Infirmary. *(p. 15)*.

LEWIS, R. B., Ph.D., M.D., Chicago, Ill. Assistant Professor, Northwestern University Medical School; Roentgenologist, Passavant Memorial Hospital. *(p. 13)*.

LICHTENSTEIN, MANUEL E., M.S., M.D., Chicago, Ill. Professor of Surgery, Cook County Graduate School; Associate Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Cook County, Michael Reese and Norwegian American Hospitals. *(p. 24)*.

LINDERT, M. C. F., B.A., B.S., M.S., M.D., Milwaukee, Wisc. Assistant Clinical Professor of Internal Medicine, Marquette University School of Medicine; Consultant in Gastroenterology, Veterans Administration Hospital. *(p. 18)*.

MADDOCK, WALTER G., M.D., M.S., Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Chairman, Department of Surgery, Chicago Wesley Memorial Hospital. *(p. 12)*.

MALKINSON, FREDERICK D., M.D., Chicago, Ill. Instructor in Dermatology, University of Chicago, School of Medicine, Albert Merritt Billings Hospital. *(p. 24)*.

MARSH, MARILYN, S.B., M.T., Miami, Fla. Supervisor, Laboratories, Parkway Medical Clinic. *(p. 18)*.

McCARTY, ROBERT T., B.S., M.D., M.S., F.A.C.G., Milwaukee, Wisc. Associate Professor of Surgery, Marquette University School of Medicine; Milwaukee County, Veterans Administration, St. Joseph's and St. Luke's Hospitals. *(p. 26)*.

McGOWAN, JOHN MALCOLM, B.S., M.D., C.M., M.S., F.A.C.S., F.R.C.S.(C), F.A.C.G., Quincy, Mass. Assistant Clinical Professor, Tufts College Medical School; Surgeon-in-Chief, Quincy City Hospital; Visiting Surgeon, Boston City Hospital. *(p. 11)*.

MELAMED, ABRAHAM, B.S., M.D., Milwaukee, Wisc. Director, Departments of Radiology, Evangelical Deaconess and St. Joseph's Hospitals. *(p. 22)*.

MILLER, GEORGE A., M.D., Urbana, Ill. Associate Roentgenologist, Carle Hospital Clinic. *(p. 15)*.

MONAT, HENRY A., A.B., M.D., F.A.C.P., F.A.C.G., Washington, D. C. Formerly Associate Clinical Professor of Internal Medicine, Georgetown University; formerly Chief of Gastroscopic Clinic, Georgetown University Hospital; formerly Chief, Gastrointestinal Department, U.S. Naval Hospital, St. Albans. *(p. 15)*.

VISIT THE EXHIBITS

MORRISON, BENJAMIN O., M.D., New Orleans, La. Instructor, Louisiana State University, School of Medicine; former Chief of Medicine, Southern Baptist Hospital; Staff Consultant, Touro Infirmary. *(p. 22)*.

MORRISON, LESTER M., M.D., F.A.C.G., Los Angeles, Calif. Assistant Professor of Medicine, College of Medical Evangelists; Senior Attending Physician, Los Angeles County Hospital; St. John's Hospital, Santa Monica. *(p. 13)*.

NECHELES, H., M.D., Ph.D., F.A.C.G., Chicago, Ill. Director, Gastrointestinal Research, Michael Reese Hospital, Professorial Lecturer, University of Chicago, Department of Physiology. *(pp. 16, 25)*.

OBERHELMAN, HARRY A., B.S., M.D., Sc.D., F.A.C.G., Chicago, Ill. Professor and Chairman, Department of Surgery, Stritch School of Medicine, Loyola University; Surgeon-in-Chief, Mercy Hospital. *(p. 14)*.

OCHSNER, ALTON, B.A., M.D., F.A.C.S., D.Sc., New Orleans, La. The William Henderson Professor of Surgery, Tulane University School of Medicine; Chairman, Department of Surgery, Tulane University; Director of General Surgery, Ochsner Clinic and Ochsner Foundation Hospital; Senior Surgeon and Chief, Tulane Surgical Service, Charity Hospital. *(p. 25)*.

OGDEN, W. W., B.S., M.D., New Orleans, La. *(p. 15)*.

PHILLIPS, KENNETH, M.S., M.D., F.A.C.G., Miami, Fla. Director, Department of Physical Medicine, Jackson Memorial Hospital. *(p. 18)*.

POLLARD, H. MARVIN, M.S., M.D., Ann Arbor, Mich. Professor of Internal Medicine, University of Michigan, School of Medicine. *(p. 13)*.

POPPER, HANS, M.D., Ph.D., Chicago, Ill. Associate Professor of Pathology, Northwestern University Medical School; Director, Department of Pathology, Cook County Hospital; Scientific Director, Hektoen Institute for Medical Research. *(pp. 11, 23)*.

POPPER, H. L., M.D., F.A.C.S., F.I.C.S., Chicago, Ill. Research Worker, Gastrointestinal Research Institute, Michael Reese Hospital. *(p. 12)*.

POSEY, E. L., Jr., B.A., M.D., M.Sc., F.A.C.G., Jackson, Miss. Attending Physician, Mississippi Baptist, St. Dominic's Hospitals; Consultant in Gastroenterology, Veterans Administration Hospital. *(p. 21)*.

POTTS, WILLIS J., M.D., Chicago, Ill. Professor of Surgery (Pediatric), Northwestern University Medical School; Surgeon-in-Chief, Children's Memorial Hospital. *(p. 25)*.

PUESTOW, KARVER L., B.S., M.D., F.A.C.P., F.A.C.G., Madison, Wisc. Professor of Medicine, University of Wisconsin, School of Medicine. *(p. 9)*.

REDEKER, ALLAN G., M.D., Los Angeles, Calif. University of Southern California, School of Medicine; Los Angeles County General Hospital. *(p. 11)*.

REYNOLDS, JOHN T., B.S., M.D., Chicago, Ill. Clinical Associate Professor of Surgery, University of Illinois College of Medicine; Attending Surgeon, St. Luke's Hospital; Associate Attending Surgeon, University of Illinois Research and Educational Hospitals. *(p. 16)*.

ROSI, PETER A., B.S., M.D., Chicago, Ill. Professor of Surgery, Cook County Graduate School of Medicine; Associate Professor, Northwestern University Medical School; Cook County, Columbus and Wesley Hospitals. *(p. 16)*.

ROSSETT, N. E., B.S., M.D., F.A.C.P., F.A.C.G., Memphis, Tenn. Teaching Gastroenterologist, St. Joseph's Hospital, Associate Physician, Methodist Hospital. *(p. 21)*.

ROTHMAN, STEPHEN, M.D., Chicago, Ill. Professor of Dermatology, University of Chicago, School of Medicine. *(p. 24)*.

RUDNER, HENRY G., SR., M.D., F.A.C.P., F.A.C.G., Memphis, Tenn. Associate Professor of Medicine, University of Tennessee, College of Medicine; Attending Physician, Baptist Memorial Hospital. *(p. 9)*.

RUDNER, HENRY G., JR., M.D., Memphis, Tenn. Assistant, Department Medical Laboratory, Division of Medicine, John Gaston Hospital and University of Tennessee; Associate Attending Physician, Baptist Memorial Hospital. *(p. 9)*.

RUKSTINAT, GEORGE J., S.B., M.D., F.A.C.G., Chicago, Ill. Clinical Professor of Pathology, Stritch School of Medicine, Loyola University; Pathologist, Holy Cross and Loretto Hospitals. *(p. 14)*.

VISIT THE EXHIBITS

SADOVE, MAX S., B.S., M.D., Chicago, Ill. University of Illinois College of Medicine; Research and Educational Hospitals; Hines Veterans Administration Hospital. (p. 17).

SARNAT, BERNARD G., M.D., F.A.C.S., Chicago, Ill. University of Illinois, Colleges of Dentistry and Medicine; Michael Reese and Woodlawn Hospitals. (p. 20).

SCHOOP, ROBERT, M.D., Chicago, Ill. Provident Hospital. (p. 21).

SCHROEDER, MORRISON, A.B., M.D., F.A.C.S., Milwaukee, Wisc. Assistant Clinical Professor of Surgery, Marquette University School of Medicine; Attending Veterans Administration Hospital. (p. 24).

SCHWIMMER, M., M.D., Brooklyn, N. Y. Chief Resident in Medicine, Metropolitan Hospital. (p. 18).

SNAPPER, I., M.D., Ph.D., F.A.C.G., Brooklyn, N. Y. Director of Medical Education, Beth-El Hospital. (p. 20).

SPAIN, DAVID M., M.D., Brooklyn, N. Y. Assistant Professor of Pathology, Columbia University, College of Physicians and Surgeons, Director of Laboratories, Beth-El Hospital. (p. 21).

SPELLBERG, M. A., B.S., M.S., M.D., F.A.C.P., F.A.C.G., Chicago, Ill. Associate Professor of Medicine, University of Illinois, College of Medicine; Attending Physician, Michael Reese and Woodlawn Hospitals; Consultant in Gastroenterology, Veterans Administration Hospital. (p. 23).

STATE, DAVID, M.D., Ph.D., F.A.C.S., Los Angeles, Calif. Assistant Clinical Professor of Surgery, University of Southern California, School of Medicine; Director of Surgery, Cedars of Lebanon Hospital. (p. 20).

STEIGMANN, FREDERICK, M.D., M.S., F.A.C.G., Chicago, Ill. Associate Professor of Medicine, University of Illinois College of Medicine; Attending Physician and Director of Therapeutics, Cook County Hospital; Attending Physician, Mt. Sinai, Columbus and Weiss Hospitals. (pp. 13, 23).

STEPHENSON, S. L., Jr., B.A., M.D., Jackson, Miss. Attending Physician, Mississippi Baptist and St. Dominic's Hospitals. (p. 21).

STRUB, IRVIN H., M.S., M.D., Chicago, Ill. Instructor in Clinical Medicine, Physician-in-Chief, Gastrointestinal Clinic, Stritch School of Medicine, Loyola University; Mercy Hospital. (p. 14).

TABERN, DONALEE L., B.S., M.S., Ph.D., Chicago, Ill. Head, Department of Radio Pharmaceuticals; Abbott Laboratories. (p. 15).

TEXTER, E. CLINTON, JR., B.A., M.D., Chicago, Ill. Associate in Medicine, Assistant Chief of Gastrointestinal Clinics, Northwestern University Medical School; Attending Physician, Passavant Memorial Hospital; Attending in Gastroenterology, Veterans Administration Research Hospital. (p. 13).

THOREK, PHILIP, M.D., B.S., F.A.C.S., F.I.C.S., Chicago, Ill. Clinical Associate Professor of Surgery, University of Illinois College of Medicine; Professor of Surgery, Cook County Graduate School of Medicine; Chief Surgeon, American Hospital; Attending Surgeon, Cook County and Alexian Brothers Hospitals. (p. 23).

VAUGHN, ARKELL M., M.S., M.D., F.A.C.S., F.A.C.G., Chicago, Ill. Clinical Professor of Surgery, Stritch School of Medicine, Loyola University; Professor of Surgery, Cook County Graduate School of Medicine; Senior Surgeon, Mercy Hospital and Free Dispensary; Attending Surgeon, Cook County Hospital. (p. 14).

VIER, HENRY J., M.D., F.A.C.S., White Plains, N. Y. Director of Surgery, St. Agnes Hospital; Consulting Surgeon, Phelps Memorial Hospital. (p. 18).

WANGENSTEEN, OWEN, H., B.A., M.D., Ph.D., Minneapolis, Minn. Professor and Chairman, Department of Surgery, University of Minnesota, School of Medicine. (p. 20).

WEINGARTEN, MICHAEL, M.D., F.A.C.G., New York, N. Y. Attending Physician, Gastroenterology, Beth Israel Hospital. (p. 10).

WEISSMAN, JACK, B.S., M.D., Brooklyn, N. Y. Clinical Assistant, Maimonides Hospital. (p. 17).

WINKELSTEIN, ASHER, M.D., B.S., New York, N. Y. Assistant Clinical Professor of Medicine, Columbia University, College of Physicians and Surgeons; Consulting Gastroenterologist, The Mt. Sinai Hospital. (p. 25).

VISIT THE EXHIBITS

BUSINESS SESSIONS

SATURDAY, 22 OCTOBER 1955

All Day
Various committee meetings at times and places to be arranged by committee chairmen.

SUNDAY, 23 OCTOBER 1955

9:00 A.M.
Meeting of the Board of Trustees—Moderne Room.

1:00 P.M.
Board of Trustees luncheon—Dubonnet Room.

3:00 P.M.
Annual Meeting of the American College of Gastroenterology—Italian Room.

6:00 P.M.
Buffet supper—Louis XVI Room.

7:30 P.M.
CONVOCATION: Presentation of Certificates—Crystal Ballroom.
See special program.

MONDAY, 24 OCTOBER 1955

4:00 P.M.
Meeting of the Credentials Committee—Dubonnet Room.

TUESDAY, 25 OCTOBER 1955

4:00 P.M.
Annual Meeting of the Board of Governors—Dubonnet Room.

WEDNESDAY, 26 OCTOBER 1955

12:30 P.M.
Luncheon meeting of the Board of Trustees—Dubonnet Room.

VISIT THE EXHIBITS

SCIENTIFIC SESSIONS

FIRST SESSION

MONDAY MORNING, 24 OCTOBER 1955

8:30 A.M. *Coffee and sweet rolls will be served in the Exhibit Area.*

LYNN A. FERGUSON, M.D., F.A.C.G., President, American College of Gastroenterology, presiding.

9:00 A.M.

1. "A Report on the Changes in Gastroenterology During the Past Fifty Years".

Speaker
DR. ISIDORE COHN, New Orleans, La.

9:20 A.M.

2. "Adrenal Influences upon the Gastrointestinal Tract".

Speaker
DR. SEYMOUR J. GRAY, Boston, Mass.

9:40 A.M.

3. "Sickle Cell Anemia with Abdominal Crisis".

Speakers
DR. HENRY G. RUDNER, SR., Memphis, Tenn. and DR. HENRY G. RUDNER, JR., Memphis, Tenn.

10:00 A.M.

4. "Role of Bacteria in the Gastrointestinal Tract".

Speaker
DR. KARVER L. PUESTOW, Madison, Wisc.

10:20 A.M.

General Discussion.

VISIT THE EXHIBITS

10:30 A.M. Recess to visit the commercial, technical and scientific exhibits.

11:00 A.M.

5. "Pathologic Anatomy of Massive Hemorrhage in Peptic Ulcer".

Speaker

DR. MILTON G. BOHROD, Rochester, N. Y. (By invitation).

11:20 A.M.

6. "Emphysematous Gastritis".

Speakers

DR. BERNARD FARFEL, Houston, Texas and DR. RALPH EICHHORN, Houston, Texas.

11:40 A.M.

7. "Nausea and Vomiting without Abdominal Pain due to Giardiasis".

Speaker

DR. MICHAEL WEINGARTEN, New York, N. Y.

12:00 Noon

General Discussion.

12:30 P.M.

LUNCHEON—Louis XVI Room. Sponsored by Burton, Parsons & Co. (admission by card only, to be obtained at the registration desk).

Speaker

DR. MARTIN E. REHFUSS, Philadelphia, Pa.

SECOND SESSION

MONDAY AFTERNOON, 24 OCTOBER 1955

ARTHUR A. KIRCHNER, M.D., F.A.C.G., Vice-President, American College of Gastroenterology, presiding.

2:00 P.M.

8. "The Present Status of Portacaval Anastomosis for Portal Hypertension".

Speaker

DR. CHARLES G. CHILD, III, Boston, Mass. (By invitation).

VISIT THE EXHIBITS

2:20 P.M.

9. "Microradiographic Studies of Hepatic Circulation".

Speaker
Dr. J. Caroli, Paris, France.

2:40 P.M.

10. "Physiological and Clinical Observations on Extrahepatic Bile Ducts".

Speaker
Dr. John M. McGowan, Quincy, Mass.

3:00 P.M.

General Discussion.

3:10 P.M. Recess to visit the commercial, technical and scientific exhibits.

3:40 P.M.

11. "Classification of Cirrhosis Based on Clinical-Pathological Correlation".

Speaker
Dr. Hans Popper, Chicago, Ill. (By invitation).

4:00 P.M.

12. "The Value of Biopsy of the Liver".

Speaker
Dr. Edward R. Christian, New Orleans, La. (By invitation).

4:20 P.M.

13. "Clinical Significance of Porphyria".

Speakers
Dr. Allan G. Redeker, Los Angeles, Calif. (By invitation) and Dr. Donald C. Balfour, Jr., Los Angeles, Calif. (By invitation).

4:40 P.M.

General Discussion.

VISIT THE EXHIBITS

THIRD SESSION

TUESDAY MORNING, 25 OCTOBER 1955

8:30 A.M. *Coffee and sweet rolls will be served in the Exhibit Area.*

> JOSEPH SHAIKEN, M.D., F.A.C.G., Secretary, American College of Gastro-enterology, presiding.

9:00 A.M.

14. "Experimental Prevention of Pancreatic Fat Necrosis".

Speaker
> DR. HANS L. POPPER, Chicago, Ill. (By invitation).

9:20 A.M.

15. "Functional Hypoglycemia".

Speaker
> DR. DALE W. CREEK, Santa Barbara, Calif.

9:40 A.M.

16. "Relapsing Pancreatitis: A Disease of Adaptation".

Speaker
> DR. MURREL H. KAPLAN, New Orleans, La.

10:00 A.M.

17. "Mechanism of Gastrointestinal Distention".

Speaker
> DR. WALTER G. MADDOCK, Chicago, Ill. (By invitation).

10:20 A.M.

General Discussion.

10:30 A.M. Recess to visit the commercial, technical and scientific exhibits.

11:00 A..M

18. "The Pathogenesis and Therapy of Human Amebiasis".

Speaker
> DR. WILLIAM W. FRYE, New Orleans, La. (By invitation).

VISIT THE EXHIBITS

11:20 A.M.

> **19. "The Effects of a Low Fat Diet on the Incidence of Gallbladder Disease".**

Speaker

> Dr. Lester M. Morrison, Los Angeles, Calif.

11:40 A.M.

> **20. "Protracted Jaundice".**

Speakers

> Dr. Frederick Steigmann, Chicago, Ill., Dr. William Dalessandro, Chicago, Ill. (By invitation) and Dr. Shibli M. Canahuati, Chicago, Ill. (By invitation).

12:00 Noon

General Discussion.

FOURTH SESSION

TUESDAY AFTERNOON, 25 OCTOBER 1955

> Frank J. Borrelli, M.D., F.A.C.G., Vice-President, American College of Gastroenterology, presiding.

2:00 P.M.

> **21. PANEL DISCUSSION ON PEPTIC ULCER**
> Dr. Clifford J. Barborka, Chicago, Ill., Moderator (By invitation).

Participants

> Dr. H. Marvin Pollard, Ann Arbor, Mich. (By invitation).
> Dr. R. B. Lewis, Chicago, Ill., (By invitation).
> Dr. E. Clinton Texter, Jr., Chicago, Ill. (By invitation).
> Dr. Walter W. Carroll, Chicago, Ill. (By invitation).

3:30 P.M.

General Discussion.

3:40 P.M. Recess to visit the commercial, technical and scientific exhibits.

VISIT THE EXHIBITS

4:10 P.M.

22. "Clinical Significance of Uropepsin".

Speaker
>   Dr. Donald C. Balfour, Jr., Los Angeles, Calif. (By invitation).

4:30 P.M.

23. "Intraabdominal Symptoms Due to Aneurysms".

Speaker
>   Dr. George J. Rukstinat, Chicago, Ill.

4:50 P.M.

24. "Experience with Vagotomy in the Treatment of Peptic Ulcer".

Speakers
>   Dr. Arkell M. Vaughn, Chicago, Ill., Dr. John M. Coleman, Chicago, Ill. (By invitation) and Dr. Irvin H. Strub, Chicago, Ill. (By invitation).

5:10 P.M.

General Discussion.

7:30 P.M.

ANNUAL BANQUET—Louis XVI Room, The Shoreland, Chicago, Ill.

FIFTH SESSION

WEDNESDAY MORNING, 26 OCTOBER 1955

8:30 A.M. *Coffee and sweet rolls will be served in the Exhibit Area.*

>   Fred H. Voss, M.D., F.A.C.G., Vice-President, American College of Gastroenterology, presiding.

9:00 A.M.

25. "Complications of Regional Enteritis".

Speaker
>   Dr. Harry A. Oberhelman, Chicago, Ill.

VISIT THE EXHIBITS

9:20 A.M.

26. "X-ray Lesions of the Small Bowel".

Speaker

Dr. L. Kenneth Beasley, St. Louis, Mo.

9:40 A.M.

27. "Anorectal Complications of Chronic Ulcerative Colitis and of Regional Enteritis".

Speaker

Dr. Emil Granet, New York, N. Y.

10:00 A.M.

28. "Complications of Abdominoperineal Resections".

Speakers

Dr. Irving A. Levin, New Orleans, La. and Dr. W. W. Ogden, New Orleans, La. (By invitation).

10:20 A.M.

General Discussion.

10:30 A.M. Recess to visit the commercial, technical and scientific exhibits.

11:00 A.M.

29. "The Use of Barium Suspended in Hydrogen Peroxide in the Radiological Study of the Esophagus and Stomach".

Speakers

Dr. Cesare Gianturco, Urbana, Ill. (By invitation) and Dr. George A. Miller, Urbana, Ill. (By invitation).

11:20 A.M.

30. "Radioisotopes of Interest in Gastroenterology".

Speaker

Dr. Donalee L. Tabern, Chicago, Ill. (By invitation).

11:40 A.M.

31. "Experience with Serpesil in Digestive Disorders".

Speaker

Dr. Henry A. Monat, Washington, D. C.

12:00 Noon

General Discussion.

VISIT THE EXHIBITS

SIXTH SESSION

WEDNESDAY AFTERNOON, 26 OCTOBER 1955

HENRY BAKER, M.D., F.A.C.G., Chairman, Board of Governors, American College of Gastroenterology, presiding.

2:00 P.M.

32. "Carcinoma of Sigmoid and Rectum".

Speaker
DR. PETER A. ROSI, Chicago, Ill. (By invitation).

2:20 P.M.

33. "Clinical and Laboratory Studies on a New Series of Spasmolytic Compounds with Specific Effects on the Gastrointestinal Tract and the Bladder".

Speaker
DR. H. NECHELES, Chicago, Ill.

2:50 P.M.

34. "Indications for Surgical Attacks on Sphincter of Oddi".

Speakers
DR. JOHN T. REYNOLDS, Chicago, Ill. (By invitation), DR. VERNON L. GUYNN, Chicago, Ill. (By invitation) and DR. BANNING G. LARY, Miami, Fla. (By invitation).

3:10 P.M. Recess to visit the commercial, technical and scientific exhibits.

3:40 P.M.

35. Ames Award Lecture.

Speaker
To be announced.

VISIT THE EXHIBITS

4:00 P.M.

36. "Caudal Anesthesia Trends".

Speaker
>Dr. Max S. Sadove, Chicago, Ill. (By invitation).

4:20 P.M.

37. "Regional Enteritis".

Speaker
>Dr. Samuel S. Berger, Cleveland, Ohio.

4:40 P.M.

General Discussion.

SEVENTH SESSION

WEDNESDAY EVENING, 26 OCTOBER 1955

Anthony Bassler, M.D., F.A.C.G., Honorary President, American College of Gastroenterology, presiding.

8:00 P.M.

38. "Errors in Anorectal Proctology".

Speaker
>Dr. John F. Keane, Boston, Mass.

8:20 P.M.

39. "Weight Control Programs".

Speaker
>Dr. S. William Kalb, Newark, N. J.

8:40 P.M.

40. "The Management of the Abdominal Anus".

Speakers
>Dr. Sidney M. Fierst, Brooklyn, N. Y., Dr. Michael Fisher, Brooklyn, N. Y. (By invitation) and Dr. Jacob Weissman, Brooklyn, N. Y. (By invitation).

VISIT THE EXHIBITS

9:00 P.M.

41. "Liver Biopsy—An Analysis of 150 Cases".

Speakers

Dr. Isidore A. Feder, Brooklyn, N. Y. and Dr. Elias Gechman, Brooklyn, N. Y. (By invitation).

9:20 P.M.

42. "Subject to be announced".

Speaker

To be announced.

9:40 P.M.

43. "Glutamic Acid in Hepatic Coma".

Speakers

Dr. Nathan W. Chaikin, New York, N. Y., Dr. Max Konigsberg, New York, N. Y. (By invitation) and Dr. M. Schwimmer, New York, N. Y. (By invitation).

10:00 P.M.

44. "Functional Behavior of the Pancreas; a Clinical Study of 1,000 Cases".

Speakers

Dr. Kenneth Phillips, Miami, Fla. and Marilyn Marsh, Miami, Fla. (By invitation).

10:20 P.M.

45. "Chronic Cholangiolitic Hepatitis".

Speaker

Dr. M. C. F. Lindert, Milwaukee, Wisc.

10:40 P.M.

46. "Acute Hemorrhagic Pancreatitis, Complicating Biliary Tract Surgery, Report of Two Fatal Cases".

Speaker

Dr. Henry J. Vier, White Plains, N. Y.

VISIT THE EXHIBITS

11:00 P.M.

47. "Nutrition and Gastrointestinal Disease".

Speaker

DR. SEYMOUR LIONEL HALPERN, New York, N. Y.

The following papers will be read by title and will appear in THE AMERICAN JOURNAL OF GASTROENTEROLOGY.

1. "Monodral in the Treatment of Gastrointestinal Disorders".
DR. ALVIN R. HUFFORD, Grand Rapids, Mich.

2. "Postcholecystectomy Syndrome".
DR. JAMES H. GOODE, Tuscaloosa, Ala.

3. "Differential Diagnosis and Treatment of Anorectal Disease".
DR. CAESAR PORTES, Chicago, Ill.

4. "Malignant Melanoma of the Rectum".
DR. ANTHONY M. SUSINNO, Palisades Park, N. J.

5. "The Previous Ligation of the Hypogastric Arteries in the Resection of Cancer of the Rectum and its Complications".
DR. R. VENANCIO TAJES, Montevideo, Uruguay.

6. "Pitfalls in the Management of Diarrheal Diseases".
DR. WILLIAM Z. FRADKIN, Brooklyn, N. Y.

COURSE IN POSTGRADUATE GASTROENTEROLOGY

SURGICAL COORDINATOR AND CO-CHAIRMAN

OWEN H. WANGENSTEEN, B.A., M.D., Ph.D., Minneapolis, Minn.

MEDICAL COORDINATOR AND CO-CHAIRMAN

I. SNAPPER, M.D., Ph.D., Brooklyn, N. Y.

FIRST SESSION

THURSDAY MORNING, 27 OCTOBER 1955

C. WILMER WIRTS, M.D., F.A.C.G., Vice-President, American College of Gastroenterology, presiding.

9:00 A.M.

Address of Welcome—
DR. JAMES T. NIX, New Orleans, La., President, American College of Gastroenterology.

9:15 A.M.

1. "Some Surgical Problems of the Oral Cavity and Related Structures".

Speaker
DR. BERNARD G. SARNAT, Chicago, Ill.

9:45 A.M.

2. "The Specificity of the Protective Role of the Pyloric Antrum in Therapy in Peptic Ulcer".

Speakers
DR. ROBERT S. KAPLAN, Los Angeles, Calif. and DR. DAVID STATE, Los Angeles, Calif.

10:15 A.M. Recess to visit commercial, technical and scientific exhibits. (Exhibits close at 2:00 P.M.)

VISIT THE EXHIBITS

10:45 A.M.

3. "Recent Experimental and Clinical Experiences with Antacid Experimentally Induced Peptic Ulceration".

Speakers
> DR. LEONIDAS H. BERRY, Chicago, Ill., DR. JONAS ADOMAVICIUS, Chicago, Ill., DR. T. J. COLE, Chicago, Ill. and DR. ROBERT SCHOOP, Chicago, Ill.

11:15 A.M.

4. "Pharmacological Background of Modern Anticholinergic Drugs".

Speaker
> DR. KAO HWANG, Chicago, Ill.

11:45 A.M.

5. "Peptic Ulcer: Medical Cure on an Ambulatory Regimen".

Speaker
> DR. N. E. ROSSETT, Memphis, Tenn.

12:15 P.M.

6. "Intestinal Carcinoid".

Speaker
> DR. DAVID M. SPAIN, Brooklyn, N. Y.

SECOND SESSION

THURSDAY AFTERNOON, 27 OCTOBER 1955

2:00 P.M.

7. "Gastritis as the Cause of Otherwise Unexplained Upper Gastro-intestinal Hemorrhage".

Speakers
> DR. E. L. POSEY, JR., Jackson, Miss. and DR. S. L. STEPHENSON, JR., Jackson, Miss.

VISIT THE EXHIBITS

2:30 P.M.

8. "Massive Hematemesis without Gross Lesion".

Speaker
      Dr. J. F. Kuzma, Milwaukee, Wisc.

3:00 P.M.

9. "Physiologic Effects of Gastrointestinal Hemorrhage".

Speaker
      Dr. Harold Laufman, Chicago, Ill.

3:30 P.M. Recess.

3:45 P.M.

10. "Radiological Aspects of Gastric Lesions Prolapsing into the Duodenal Bulb".

Speaker
      Dr. Abraham Melamed, Milwaukee, Wisc.

4:15 P.M.

11. "Hereditary Telangiectasia Complicated by a Duodenal Ulcer".

Speaker
      Dr. Benjamin O. Morrison, New Orleans, La.

THIRD SESSION

FRIDAY MORNING, 28 OCTOBER 1955

9:00 A.M.

12. "Clinical Guides to Diagnosis of Jaundice".

Speaker
      Dr. Herman F. DeFeo, Chicago, Ill.

VISIT THE EXHIBITS

9:30 A.M.

13. "Experiences with Needle Biopsy of the Liver".

Speakers
>Dr. FLOYD M. BEMAN, Columbus, Ohio, DR. C. JOSEPH DELOR, Columbus, Ohio and DR. DAVID BROWN, Columbus, Ohio.

10:00 A.M.

14. "Therapeutic Considerations in Acute and Chronic Diseases of the Liver".

Speaker
>DR. FREDERICK STEIGMANN, Chicago, Ill.

10:30 A.M. Recess.

11:00 A.M.

15. "Inflammatory Diseases of the Pancreas".

Speaker
>DR. PHILIP THOREK, Chicago, Ill.

11:30 A.M.

16. "The Usefulness of Corticotropin and Corticoids in Patients with Liver Disease".

Speaker
>DR. MITCHELL A. SPELLBERG, Chicago, Ill.

12:00 Noon

BUFFET LUNCHEON (Admission by Card only)

FOURTH SESSION

FRIDAY AFTERNOON, 28 OCTOBER 1955

1:00 P.M.

CLINICAL-PATHOLOGICAL CONFERENCE
Moderator: DR. HANS POPPER, Chicago, Ill.

VISIT THE EXHIBITS

2:30 P.M.

17. "Changes in the Gastrointestinal Tract in Scleroderma and other Diffuse Connective Tissue Diseases".

Speakers
> DR. STEPHEN ROTHMAN, Chicago, Ill. and DR. FREDERICK D. MALKINSON, Chicago, Ill.

3:00 P.M. Recess.

3:30 P.M.

18. "Ulcerative Colitis—Current Concepts".

Speaker
> DR. JOSEPH B. KIRSNER, Chicago, Ill.

4:00 P.M.

19. "Indications for Colostomies".

Speaker
> DR. MANUEL E. LICHTENSTEIN, Chicago, Ill.

4:30 P.M.

20. "Colectomy During Acute Phase of Chronic Ulcerative Colitis".

Speaker
> DR. MORRISON SCHROEDER, Milwaukee, Wisc.

FIFTH SESSION

SATURDAY MORNING, 29 OCTOBER 1955

9:00 A.M.

21. "Indications for Pyloroplasty, Vagotomy, Gastroenterostomy and Resection".

Speaker
> DR. WALTER C. BORNEMEIER, Chicago, Ill.

VISIT THE EXHIBITS

9:30 A.M.

22. "Effects of Hormones on Secretion of Pepsin".

Speaker
> Dr. Irving B. Fritz, Chicago, Ill.

10:00 A.M.

23. "Gastrointestinal and Urinary Spasmolytic Drugs".

Speakers
> Dr. H. Necheles, Chicago, Ill. and Dr. N. C. Jefferson, Chicago, Ill.

10:30 A.M. Recess.

11:00 A.M.

24. "Classification and Treatment of Different Varieties of Peptic Esophagitis".

Speaker
> Dr. Asher Winkelstein, New York, N. Y.

11:30 A.M.

25. "Carcinoma of the Stomach: Need for Earlier Diagnosis".

Speakers
> Dr. Alton Ochsner, New Orleans, La. and Dr. John Blalock, New Orleans, La.

SIXTH SESSION

SATURDAY AFTERNOON, 29 OCTOBER 1955

2:00 P.M.

26. "Gastrointestinal Anomalies in Children".

Speaker
> Dr. Willis J. Potts, Chicago, Ill.

VISIT THE EXHIBITS

2:30 P.M.

27. "Surgical Management of Regional Ileitis".

Speaker
     Dr. A. R. Curreri, Madison, Wisc.

3:00 P.M.

28. "Diagnosis and Treatment of Polyps of the Rectum and Colon".

Speaker
     Dr. Robert T. McCarty, Milwaukee, Wisc.

SCIENTIFIC EXHIBITS

BOOTH S-1 **"Ulcerative Colitis and Familial Polyposis—Surgical Management and Value of One-Stage Total Colectomy".**

DR. LOUIS T. PALUMBO and DR. GEORGE M. RUGTIV, Des Moines, Iowa.

The exhibit presents a newer concept to the surgical approach in the management of patients with chronic ulcerative colitis and familial polyposis. It portrays the indication for surgery, the incidence of carcinoma, complications of the disease, and the roentgenographic findings. It presents the various types of surgical procedures performed and the method and care of the ileostomy as well as the comparative mortality and morbidity rates of the various surgical procedures. The advantages of a one-stage total colectomy (including the abdominoperineal portion) with or without a preliminary ileostomy are presented and the technic of this procedure is portrayed.

BOOTH S-2 **"Recent Experimental and Clinical Experiences with Antacid Therapy in Peptic Ulcer".**

DR. LEONIDAS H. BERRY, DR. ROBERT SCHOOP and DR. DAVID FELDMAN, Chicago, Ill.

The exhibit is designed to show clinical and laboratory experiences with Dihydroxy-aluminum-aminoacetate as an antacid. There will be curves indicating the neutralizing value of the chemical in cases of gastric, duodenal and gastrojejunal ulcer and hypertrophic gastritis. There will be curves of a control group showing neutralization in patients without gastro-intestinal diseases. Acid-antacid relationships will be shown in terms of titration values with 0.1 normal NaOH and pH values determined with an electric pH meter. Correlation of all data with age, sex, and other variables and clinical course of the disease.

Further correlation studies were made by gastroscopic examinations and repeated follow-up by x-ray and gastroscopy to follow the course of healing, especially in gastric ulcer.

BOOTH S-3 **"Polyps of Rectum and Colon".**

DR. ROBERT T. McCARTY, Milwaukee, Wisc.

This exhibit is in correlation with a paper which is being presented for the postgraduate course. It will show the distribution of polyps, pictures of unusual polyps, and models of four buttocks with sigmoidoscope inserted through the anus showing models of polyps and carcinoma.

BOOTH S-4 **"Ambulatory Continuous Drip Therapy in Peptic Ulcer".**

DR. FREDERICK STEIGMANN, Chicago, Ill.

Drawings showing the importance of gastric acidity in the genesis and persistence of peptic ulcer are presented; similarly charts and graphs showing the incomplete and short duration of neutralization of the gastric acidity by presently used antacids. The value of the original Winkelstein drip method in more complete and prolonged neutralization of gastric acidity in bed patients is discussed and a new method of ambulatory continuous drip therapy demonstrated and its clinical application presented.

BOOTH S-5 **"Anatomy of the Pancreaticoduodenal Complex".**
DR. WILLIAM P. KLEITSCH, Omaha, Neb.

The exhibit consists of a description of the anatomy of the pancreatic duct system. The embryology of the pancreas is reviewed and its significance in the adult pancreatic duct anatomy is explained. Photographs of representative variations of the normal duct arrangement are shown. The clinical significance of the arrangement of the pancreatic ducts relative to gastric surgery is indicated and the normal arrangement of the blood supply to the pancreas is described. In addition to the photographs and legends, representative specimens of those studied will be available for examination.

BOOTH S-6 **"Tube Feeding".**
DR. JAMES BARRON, DR. JAMES L. SAWYER and DR. LAWRENCE
S. FALLIS, Detroit, Mich.

The methods of preparing liquefied natural foods are shown and small feeding pumps are demonstrated. Emphasis is placed on the practical aspects for use in the home and in small hospitals. Natural whole foods are inexpensive and quite well tolerated. The tubes used are of fine caliber, inexpensive and easily inserted by the use of technics to be demonstrated. Some follow-up charts will be available as will quantitative food analyses.

BOOTH S-7 **"The Medicinal Management of Fatty Alcoholic Livers".**
DR. THADDEUS D. LABECKI and DR. CARROLL L. BUSBY,
Jackson, Miss.

The exhibit will consist of photomicrographs demonstrating to what extent the fatty infiltration in Laennec's cirrhosis may be alleviated as evidenced by punch biopsy. The description of regimens investigated and the conclusions drawn from the study, which included patients followed for up to two years, are included. Some of the patients were first subjected to the conventional nutritional management and subsequently treated with synergistically acting lipotropic agents. Others were followed for varying periods of time on lipotropic therapy only or on a diet containing up to 50 grams of protein per day with moderately large doses of lipotropic substances. Parallel to the biopsy studies, response to nutritional and medicinal management has been gauged with the aid of various liver function tests (cephalin flocculation, BSP retention, thymol turbidity, serum cholesterol, protein profile). Correlation, or lack of it, between morphological studies and the so-called function tests is emphasized.

TECHNICAL EXHIBITORS

(Those attending the Convention sessions are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with the many new products and new equipment on display.)

AMES COMPANY, INC., Elkhart, Ind. (Booth 9). Representatives Messrs. Bayard E. Simmons and John E. Bullock, Jr. will be on hand to discuss *Aminet* and *Diatussin*. *Aminet* combines Aminophylline and Pentobarbital in a newly developed nonreactive base which melts readily at body temperatures and quickly releases the active ingredients for rapid absorption. *Diatussin*, nonnarcotic antitussive especially effective in cough accompanying laryngitis, pharyngitis, bronchitis, pertussis, or the common cold.

THE BORDEN COMPANY, New York, N. Y. (Booth 6).

BRISTOL LABORATORIES, New York, N. Y. (Booth 15). Available at the booth—literature and samples on *Centrine*—a potent synthetic antispasmodic, anticholinergic. Also information on *Kectil*, an adjunct in the treatment of ulcerative colitis and regional enteritis during the acute active stages of the disease.

BURROUGHS WELLCOME & CO., INC., Tuckahoe, N. Y. (Booth 13). The extensive research facilities of 'B. W. & Co.' both here and in England are directed to the development of improved therapeutic agents and technics.

Through such research 'B. W. & Co.' has made notable advances related to cancer, malaria, diabetes, antibiotics, muscle relaxants, and autonomic, antihistaminic, and antinauseant drugs. An informed staff will be at the booth to discuss the products and latest developments.

BURTON, PARSONS & COMPANY, Washington, D. C. (Booth 22). You are cordially invited to visit this booth where information, samples and literature will be available on the hydrophilic colloids, L. A. Formula and Konsyl. Orange juice will also be available to prove that the L. A. Formula is unsurpassed for palatability and literally defies detection in citrus juices.

CAMERON SURGICAL SPECIALTY COMPANY, Chicago, Ill. (Booth 14). Visit the interesting and informative exhibit. The Cameron Company developed the first American made gastroscope and now offers its patented Omniangle scope with coated lenses which approximately doubles the field of vision. Also on display will be the Plastic Model of the Human Stomach, Cavicamera, Boros Flexible Esophagoscope and other items of interest to the Gastroenterologist.

THE COCA-COLA CO., Atlanta, Ga. (Lounge). Ice-cold Coca-Cola served through the courtesy and cooperation of the Coca-Cola Bottling Company of Chicago, Inc., and the Coca-Cola Company.

EDER INSTRUMENT COMPANY, Chicago, Ill. (Booth 16), will again exhibit their latest developments in gastroscopic equipment. The Eder-Hufford Flexible Esophagoscope is happy to announce the arrival of two new additions—two little baby gastroscopes small enough to be introduced through the Eder-Hufford Esophagoscope. Be sure to see them.

ENCYCLOPEDIA BRITANNICA, Chicago, Ill. (Booth 20).

VISIT THE EXHIBITS

MALLON CHEMICAL CORPORATION, New York, N. Y. (Booth 23), subsidiary of the DOHO CHEMICAL CORPORATION, makers of *Auralgan*, *New Otosmosan*, and *Rhinalgan*, are pleased to exhibit *Rectalgan*, the liquid topical anesthesia for relief of pain and itching in hemorrhoids and pruritus; following perineal suturing in obstetrical and gynecological work, and for many other uses in pre- and postoperative cases.

MALTBIE LABORATORIES DIVISION WALLACE & TIERNAN INC., Belleville, N. J. (Booth 27). All physicians are cordially invited to visit the exhibit. Featured items will be: *Desenex*, the well-known undecylenate fungicide; *Malcotran*, high potency, low dosage anticholinergic; *Cholan-HMB*, for biliary dysfunction; and *Calpurate*, our myocardial stimulant, vasodilator and mild diuretic. Representatives will welcome the opportunity to discuss these preparations.

F. MATTERN MANUFACTURING COMPANY, Chicago, Ill. (Booth 25), presents a complete Radiographic, Fluoroscopic, Diagnostic Unit featuring Trendelenburg Tilt Table and Spot Film Device. Personnel are on hand to discuss X-ray equipment and technical problems.

THE NATIONAL DRUG COMPANY, Philadelphia, Pa. (Booth 17), will feature the *Resions* offering two effective compounds for treatment of almost any diarrheal condition found in clinical practice. The *Resions* act by ion exchange . . . to attract, bind and remove toxic materials in diarrheas caused by food or bacterial toxins, by prolonged use of certain drugs, and in general infectious diseases. The *Resions* are safe because they are totally insoluble and nontoxic. Resion therapy will control about 90% of common diarrheas. Resion P-M-S is intended especially for rapid control of those rare diarrheas caused by Gram-negative organisms; to prevent secondary bacterial infection; in mycotic diarrhea following the use of the broad-spectrum antibiotics, and to inhibit the enteric growth of C. albicans (Monilia).

PET MILK COMPANY, St. Louis, Mo. (Booth 1). Representatives will be on hand to discuss the merits of the new *Instant* 'Pet' Nonfat Dry Milk. They will be pleased to have you stop and discuss its many uses. Literature will be mailed to your office upon request.

THE PURDUE FREDERICK COMPANY, New York, N. Y. (Booth 5), will present *Senokot*, a new standardized large-bowel neuroperistaltic for controlled constipation correction. A botanical (percarp fraction of Cassia acutifolia), it acts specifically to stimulate Auerbach's plexus and simulate normal peristaltic rush and defecatory reflex. The active glycosides are inert and non-irritating in stomach and small bowel.

WILLIAM H. RORER, INC., Philadelphia, Pa. (Booth 28), presents *Probutylin* (Procaine Isobutyrate–Rorer) a new, clinically proven drug for control of nausea, vomiting, gastritis and many other undesirable reflex manifestations of gastrointestinal origin. *Maalox* (magnesium aluminum hydroxide), a nonconstipating, pleasant tasting antacid for peptic ulcer, gastritis and heartburn in pregnancy, will also be featured.

SANDOZ PHARMACEUTICALS, Hanover, N. J. (Booth 21). *Cafergot*–Available in oral and rectal form for effective control of head pain in migraine and other vascular headaches.

Bellegral–A time-tested preparation for use in functional disorders.

Plexonal–A new sedative-hypnotic. *Plexonal* is not just another hypnotic, but is one developed on a new pharmacologic approach. The action of subthreshold doses of classic sedative agents are potentiated and enhanced by autonomic and central acting drugs.

Belladenal–The most potent natural antispasmodic sedative for the control of hypermotility with pain and hypersecretion of the intestinal tract.

VISIT THE EXHIBITS

SCHENLEY LABORATORIES, INC., New York, N. Y. (Booth 10), will feature:

MorCal—new appetizing high calorie food supplement containing Vitamins B₁ and B₁₂ for quick weight gain and energy.

Ediol—a palatable oral fat emulsion for quick gain in weight and energy.

Titralac—an effective antacid because it titrates like milk.

Sedamyl—a nonbarbiturate, ideal for daytime sedation without drowsiness.

Dorbane—a precise potency for individualized dosage in the treatment of constipation.

SCHERING CORPORATION, Bloomfield, N. J. (Booth 18). Members of the American College of Gastroenterology and their guests are cordially invited to visit the Schering exhibit where new therapeutic developments will be featured.

Schering representatives will be present to welcome you and to discuss with you these products of our manufacture.

SMITH, KLINE & FRENCH LABORATORIES, Philadelphia, Pa. (Booth 8), is proud to present its line of specialties, including 'Prydon' and 'Prydonnal' *Spansule* sustained release capsules for prolonged anticholinergic activity in peptic ulcer, and spastic conditions of the gastrointestinal tract. We welcome the opportunity to answer your questions at our booth.

E. R. SQUIBB & SONS, New York, N. Y. (Booth 7). New Squibb Products, and new brochures of useful interest to you on products already introduced, will be featured.

As in former years, your Squibb Representative again cordially invites you to visit the Squibb Booth.

WINTHROP-STEARNS INC., New York, N. Y. (Booth 26). *Monodral* bromide (5 mg.) with *Mebaral* (½ grain), well tolerated anticholinergic sedative for tense ulcer patients with peptic ulcer (gastric and duodenal), hyperacidity, gastritis, pylorospasm, gastrointestinal motility, spastic and irritable colon. Causes little or no drowsiness and is relatively free from other troublesome side-effects. And *Creamalin*, nonalkaline, nonabsorbable antacid.

VISIT THE EXHIBITS

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In Memoriam

We record with profound sorrow the passing of Dr. Patrick M. Moriarty, Associate Fellow, of Chicopee, Mass. We extend our deepest sympathies to the bereaved family.

"Ulcer pain and other symptoms disappear rapidly"[1]

PRANTAL *

anticholinergic

with outstanding freedom

from side effects[2]

Individualized therapy of peptic ulcer, pyloro-spasm, gastric hyperacidity and hypermotility, and chronic hypertrophic gastritis.

For easily adjusted dosage . PRANTAL Tablets
For eight hours' relief
     with single dose . . . . . PRANTAL REPETABS
For acute episodes . . . . . . PRANTAL Injection

1. Hoffmann, C. R.: Am. Pract. & Digest Treat.
   4:464, 1953.
2. Riese, J. A.: Am. J. Digest. Dis. *21*:81, 1954.

PRANTAL® Methylsulfate, brand of diphemanil
methylsulfate.

REPETABS,® Repeat Action Tablets.

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for the "Sippy-diet" patient

a welcome *(and often necessary)* change from "milk-and-cream"

MULL-SOY® Powdered

Pioneer soy alternative to milk... reported to be "noticeably more soothing to the upper gastrointestinal tract and seemingly easier to digest."[1] Comparable to milk in buffering[2] and nutritional[3] qualities. Contains no cholesterol ... and costs the patient *much* less than milk-and-cream. Easy to prepare — 4 level tablespoonfuls to 8 oz. water. In 1-lb. tins at all drug outlets.

1. Balfour, D. C., Jr.: Am. J. Gastroenterol. 22:181, 1954.
2. Burke, J. O., et al.: Internat. Rec. Med. & Gen. Practice Clin. 167:587, 1954. 3. Sternberg, S. D., and Greenblatt, I. J.: Ann. Allergy 9:190, 1951.

Are you wondering how MULL-SOY Powdered tastes? Return this coupon for professional trial samples and see for yourself how *pleasant* it can be for your milk-weary or milk-intolerant ulcer patients.

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Please send to me, without charge, four 4-oz. tins of MULL-SOY Powdered.

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Mr. Daniels
was willing to
be bombed
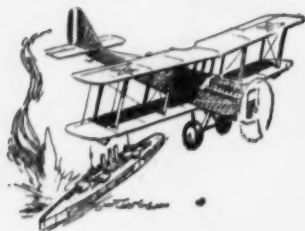
ADMIRALS smiled when, in 1921, he claimed air power could sink battleships. And Mr. Josephus Daniels, the Navy secretary, said he was "prepared to stand bareheaded on the deck of a battleship and let General Mitchell take a crack at me with bombing airplane."

But in an actual test, the most heavily armored dreadnaught ever built sank in minutes under the sledge-hammer blows of the world's first 1-ton bombs—bombs built to Billy Mitchell's order.

Mitchell was used to disbelief. In World War I, Pershing called his idea for dropping infantry by parachute absurd. "Experts" laughed when he talked of putting cannon in planes, scoffed when he predicted air speeds way in excess of 200 miles.

In his early fight for a strong air force, Mitchell saw very dark days. Yet he never lost faith in the American people, nor they in him. For they recognized his clear foresight and great fighting heart as part of the real American spirit.

It is this courageous spirit that makes America strong—so strong, in fact, that our country's Savings Bonds are regarded as one of the finest investments in the world.

Why not take advantage of that strength? Use United States Savings Bonds to guard your future, and your country's future. Invest in them regularly—and hold onto them.

It's actually easy to save money—when you buy United States Series E Savings Bonds through the automatic Payroll Savings Plan where you work! You just sign an application at your pay office; after that your saving is done for you. And the Bonds you receive will pay you interest at the rate of 3% per year, compounded semiannually, for as long as 19 years and 8 months if you wish! Sign up today! Or, if you're self-employed, invest in Bonds regularly where you bank. There's no surer place to put your money, for United States Savings Bonds are as safe as America!

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VISIT THE EXHIBITS

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Ulcer protection that lasts all night:

Pamine*-Phenobarbital
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Tablets

Each FULL-STRENGTH tablet contains:

Phenobarbital _____ 15.0 mg. ($\frac{1}{4}$ gr.)
Methscopolamine bromide _____ 2.5 mg.

Dosage:

One tablet one-half hour before meals, and 1 to 2 tablets at bedtime.

Each HALF-STRENGTH tablet contains:

Phenobarbital _____ 8.0 mg. ($\frac{1}{8}$ gr.)
Methscopolamine bromide _____ 1.25 mg.

Dosage:

While the dosage and indications are the same as for the full-strength tablets, this tablet allows greater flexibility in regulating the individual dose, and may be employed in less severe gastrointestinal conditions.

Supplied:

Both strengths in bottles of 100 tablets.

*REGISTERED TRADEMARK FOR THE UPJOHN BRAND OF METHSCOPOLAMINE

The Upjohn Company, Kalamazoo, Michigan

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"A neglected diagnostic procedure".*...now simplified with...

THE **FLEET ENEMA**

DISPOSABLE UNIT

Because the fever of infection increases metabolic needs

Tetracyn SF*

Brand of tetracycline

is indicated · to combat the invading organism with TETRACYN, the newest and best-tolerated broad-spectrum antibiotic

· to support the patient's natural defenses with the vitamins essential for resistance and recovery

with a single prescription. This concept, originated by Pfizer,

results in · maximum antibiotic blood levels[1]

· superior clinical effectiveness[2]

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Terramycin† SF* is a similar combination; the average daily dose (1 Gm.) of Tetracyn or Terramycin supplies the needed amount of a special vitamin formula recommended by Pollack and Halpern[4] for nutritional therapy of patients under physiological stress.

Supplied: TETRACYN SF, Capsules 250 mg.; Oral Suspension (fruit flavored) 125 mg./5 cc. teaspoonful.
TERRAMYCIN SF, Capsules 250 mg.

1. Dumas, K. J.; Carlozzi, M., and Wright, W. A.: Antibiotic Med. *1:*296 (May) 1955. 2. Prigot, A.: Ann. New York Acad. Sc., in press. 3. Milberg, M. B., and Michael, M., Jr.: Ibid. 4. Pollack, H., and Halpern, S. L.: Therapeutic Nutrition, Prepared in Collaboration with the Committee on Therapeutic Nutrition, Food and Nutrition Board, National Research Council, Washington, D. C., 1952.

*Trademark for Pfizer brand of antibiotics with vitamins.
†Brand of oxytetracycline

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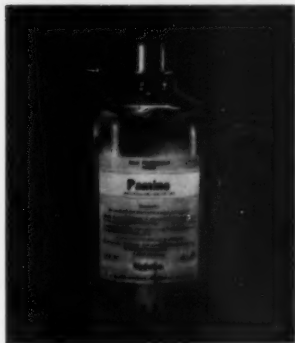
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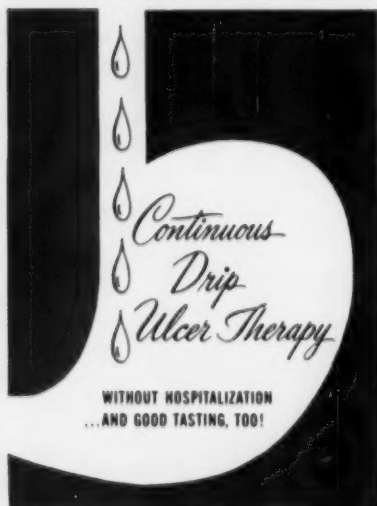
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SULFASUXIDINE® SUSPENSION WITH PECTIN AND KAOLIN

MAJOR ADVANTAGES: Has pronounced antibacterial action. Detoxifies and adsorbs intestinal irritants. Soothes the mucosa. Tasty chocolate-mint flavor.

Adult dosage: 1½ to 2 tbsp. six times a day. *Children and infants* in proportion.

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DIVISION OF
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Continuous Drip Ulcer Therapy

WITHOUT HOSPITALIZATION
...AND GOOD TASTING, TOO!

Nulacin

A recent clinical study* of 46 ambulatory non-hospital patients treated with Nulacin† and followed up to 15 months describes the value of ambulatory continuous drip therapy by this method. Total relief of symptoms was afforded to 44 of 46 patients with duodenal ulcer, gastric ulcer and hypertrophic gastritis.

The delicately flavored tablets dissolve slowly in the mouth (not to be chewed or swallowed). They are not noticeable and do not interfere with speech.

Nulacin tablets are supplied in tubes of 25 at all pharmacies. Physicians are invited to send for reprints and clinical sample.

*Steigmann, F., and Goldberg, E.: Ambulatory Continuous Drip Method in the Treatment of Peptic Ulcer, Am. J. Digest. Dis. *22*:67 (Mar.) 1955.

†Mg trisilicate 3.5 gr.; Ca carbonate 2.0 gr.; Mg oxide 2.0 gr.; Mg carbonate 0.5 gr.

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Pharmaceutical Division

RACINE, WISCONSIN

Greater acid-binding capacity[1]

Longer therapeutic effect[1]

A comparative test shows conclusively that Maalox
not only has more than double the acid-binding
capacity of aluminum hydroxide, but also maintains its
effectiveness twice as long.[1]

Another investigator emphasizes that "Maalox is
preferable to aluminum hydroxide gel because it is
more palatable, better tolerated by the stomach, and
does not cause constipation or undue astringency."[2]

Maalox-Rorer is a well-balanced suspension of
magnesium aluminum hydroxide gel—smooth-textured
and pleasant to taste. It enjoys unusual patient
acceptance.

MAALOX ®

"...better suited for antacid therapy"[2]

SUPPLIED: Suspension, bottles of 12 fluid ounces.
Tablets, bottles of 100.

Samples sent promptly on request

RORER

WILLIAM H. RORER, INC.

PHILADELPHIA 6, PA.

1. Rossett, N.E., Rice, M.L. Jr.: An In Vitro Evaluation of the More Frequently Used Antacids, Gastroenterology
26:490 (1954).
2. Morrison, Samuel: Magnesium Aluminum Hydroxide Gel in the Antacid Therapy of Peptic Ulcer, Am. J.
Gastroenterology 22:309 (1954).

SEE OUR EXHIBIT AT BOOTH 28

Upjohn

Ulcer protection
that
<u>lasts all night:</u>

Pamine tablets

Bromide REGISTERED TRADEMARK FOR THE UPJOHN BRAND OF METHSCOPOLAMINE

Each tablet contains:
Methscopolamine bromide
                                    2.5 mg.

Average dosage (ulcer):
One tablet one-half hour before
meals, and 1 to 2 tablets at
bedtime.

Supplied:
Bottles of 100 and 500 tablets.

The Upjohn Company, Kalamazoo, Michigan
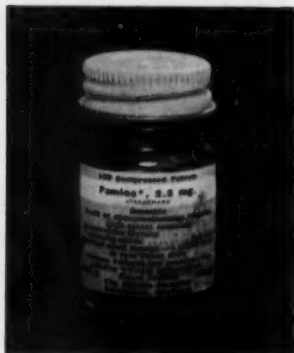
In bulk therapy . . .

palatability is often the determining factor between success and failure

The effectiveness of any hydrophilic colloid is directly proportional to its palatability—all other things being equal. Elementary though it may be, the principle bears repeating that no medication will work if your patient won't take it.

L. A. FORMULA, the original refined hydrophilic colloid, is unsurpassed for effectiveness. It is pleasant to take in cool water—literally defies detection in milk or the popular citrus juices. And although L. A. Formula goes into solution immediately it takes up to 10 minutes to gel. This allows even your slowest patient ample time in which to drink this smooth mixture.

Combine with palatability a noteworthy degree of effectiveness[1,2] and a significantly lower cost-to-patient and you have a preparation that demands investigation. Send for samples of L. A. Formula today. You'll approach your cases of chronic constipation with a new confidence once you have tried this preparation.

1. *Cass & Wolf: Gastroenterology 20:149, 1952*

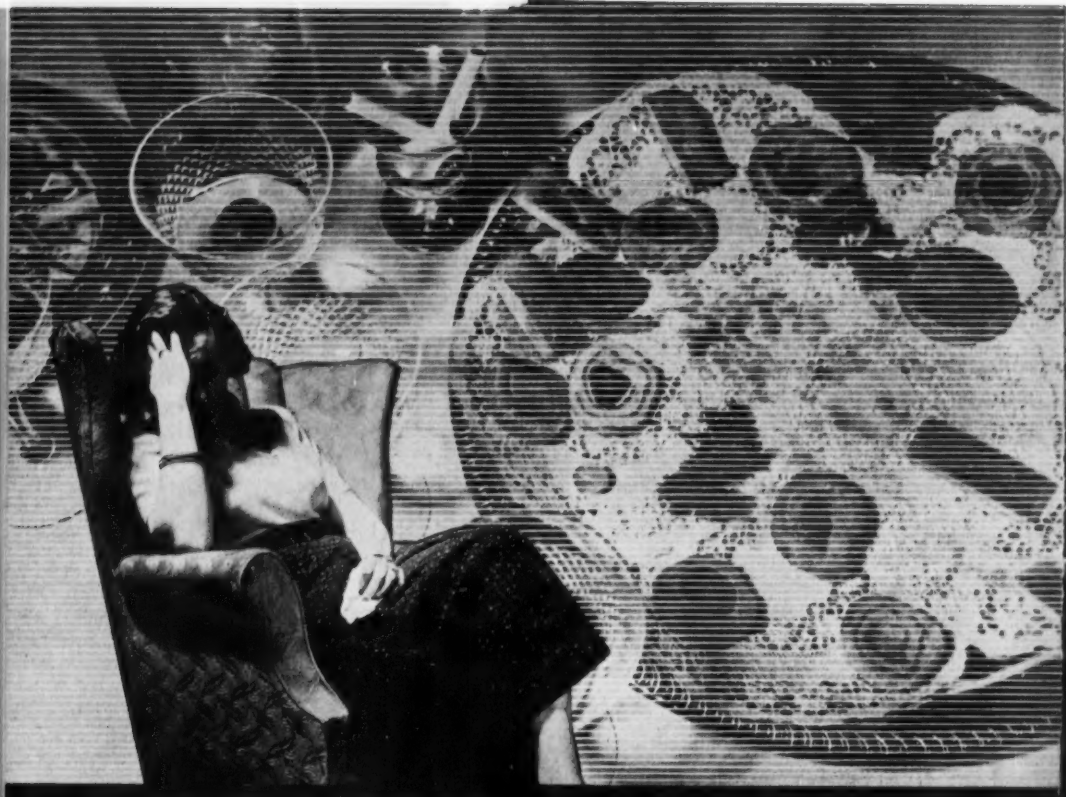**2.** *Cantor: Am. J. Proctol. 3:204, 1952*

FORMULA: 50% Plantago ovata dispersed in lactose and dextrose. AVAILABLE: 7 and 14 ounce containers at $1.25 and $2.25 respectively.

Burton, Parsons & Company
Washington 9, D. C.

L. A. Formula

Gastric Hyperacidity: etiology

People being people, environmental factors contributory to gastric hyperacidity are hard to remove, even when their role is clearly defined. But, the physician has a sure, simple—even pleasant—way of relieving the acid distress caused by:

- dietary indiscretion
- nervous tension
- emotional stress
- food intolerances
- excessive smoking
- alcoholic beverages

Gelusil promptly and effectively controls the excessive gastric acidity of "heartburn" and chronic indigestion. And it affords equally rapid relief in peptic ulcer. Sustained action is assured by combining magnesium trisilicate with the specially prepared aluminum hydroxide gel.

Free from constipation: Gelusil's aluminum hydroxide component is specially prepared: the concentration of aluminum ions is accordingly low; hence the formation of astringent—and constipating—aluminum chloride is minimal.

Free from acid rebound: Unlike soluble alkalies, Gelusil does not over-neutralize or alkalinize. It maintains the gastric pH in a mildly acid range—that of maximum physiologic functioning.

Dosage—2 tablets or 2 teaspoonfuls two hours after eating or when symptoms are pronounced. Each tablet or teaspoonful provides: $7\frac{1}{2}$ gr. magnesium trisilicate and 4 gr. aluminum hydroxide gel.

Available — Gelusil Tablets in packages of 50, 100, 1000 and 5000. Gelusil Liquid in bottles of 6 and 12 fluidounces.

Gelusil®

Antacid · Adsorbent

WARNER-CHILCOTT